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AN INTRAPERICARDIAL TERATOMA AND A TUMOR OF THE HEART: BOTH REMOVED OPERATIVELY

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I should like to report two lesions of the heart and pericardium. The patients presenting these lesions were referred to me by Dr. Harold Feil, to whom I am indebted because they form the basis of this report.

INTRAPERICARDIAL TERATOMA

Case 1.—This patient was a white male, age 22. Complaints when admitted to the University Hospitals, June 25, 1934, were fever, weakness and pain in the chest. The patient had been well until February, 1932, when his symptoms first appeared. He was admitted to the Mt. Sinai Hospital, Cleveland, in October, 1932. At that time the cardiopericardial silhouette was enormously enlarged (Fig. 1), and 280 cc. of bloody fluid were removed from the pericardial cavity by aspiration. The fluid was examined for tubercle bacilli but none were found. Signs of severe cardiac compression were present. Cardiac compression was relieved by the removal of fluid but recurred after each aspiration. The temperature fluctuated between 38° and 39.5° C. Improvement occurred during the month in the hospital but after one month at home he was readmitted because of the recurrence of symptoms. Aspiration (presumably of the pericardial cavity) yielded 500 cc. of chocolate-colored fluid. A large, encapsulated, cyst-like outpouching of the pericardium appeared, with a smaller encapsulation just above it (Fig. 2). The area of encapsulation was punctured by a needle but nothing was obtained. The patient had episodes of fever in 1933, but then improved and was at school during the first half of 1934. In June he developed fever again and was admitted to this hospital. The temperature was 38° to 39° C. The venous pressure in the arm was 14 cm. of water. The arterial pressure was 110/70 mm. mercury. Cyanosis, shortness of breath, palpitation of the heart and enlargement of the liver, which had been present intermittently in the past, were not present when admitted to the hospital. Roentgenogram of the chest was about the same as in Figure 2. A slight precordial bulge was present to the right of the sternum.

First Operation.—The fourth right costal cartilage was removed. A structure which was considered to be parietal pericardium was incised. It was 3 to 4 mm. in thickness. This incision opened a cyst containing 300 cc. of material which looked like water mixed with coagulated yolk of egg. The cyst was walled-off from the general pericardial cavity, which was not opened. At the base of the cyst the contour of the right auricle and the right ventricle was seen. The cyst was emptied and washed with solution of sodium chloride. The margin of the incision in the cyst was sutured to the pectoral muscle and the wound was loosely closed without drainage. Some

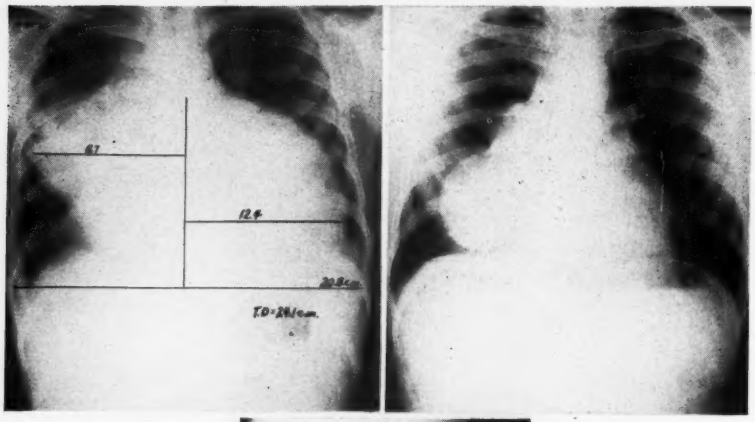


FIG. 1. — Cardiopericardial silhouette is greatly enlarged. Bloody fluid was removed by aspiration. The heart was compressed (October, 1932).

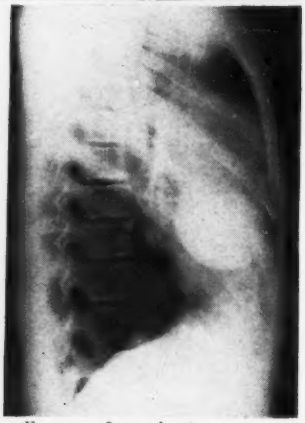


FIG. 2.—Roentgenogram taken January, 1933. Chocolate-colored fluid, 500 cc., removed by aspiration. Cyst-like outpouching of pericardium, with a smaller mass just above it developed on the right side.

FIG. 3.—Lateral view of chest showing mass along right border of the heart (1936).

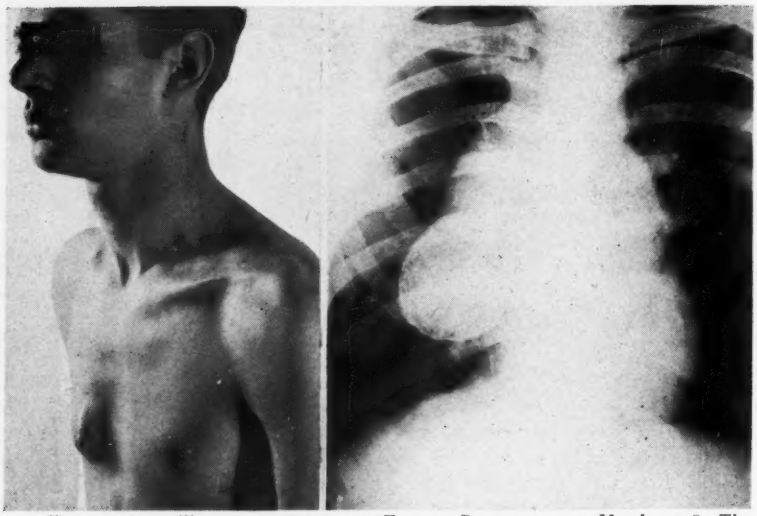


FIG. 4.—A swelling appeared to the right of the sternum (March, 1938).

FIG. 5.—Roentgenogram, March, 1938. The wall of the cyst shows deposition of calcium and some reduction in size.

fluid seeped from the wound into the dressings for three days, but the wound healed and there was no further drainage. Examinations of smears of the material were reported to have shown bacteria but none were grown and guinea-pig inoculation did not show tuberculosis.

For the next two years the patient was improved, then his symptoms recurred. Doctor Feil reported that, in September, 1936, the venous pressure in the arm was 21 cm. of water. Lateral view of the chest showed a mass extending along the right ventricle, right auricle and vena cava (Fig. 3). This episode of cardiac compression was transient and he again improved until March, 1938, when he developed a swelling in the region of the operative scar (Fig. 4). He was readmitted to the hospital. The temperature was normal. The venous pressure was 12 cm. of water. No ascites. No enlargement of the liver. Roentgenogram of the chest is shown in Figure 5.

Second Operation.—The scar of the original operation was excised and the mass was opened. Hair was found in the contents of the cyst. The cyst contained 350 cc. of yellow fluid and masses of amorphous material. The cyst was emptied and its lining was washed with a solution of sodium chloride. In the base of the cyst the outline of aorta and pulmonary artery was seen. The wall of the cyst was then excised by sharp dissection. It was dissected from aorta, pulmonary artery, vena cava, right ventricle and right inferior pulmonary vein. It was densely adherent in the region of the great transverse sinus. At one time, while the dissection was carried out over the superior vena cava, the patient suddenly stopped breathing but started again after about one minute. Silver clips, ligatures and transfixion sutures were used for hemostasis. The cyst was completely removed (Fig. 6). I had the impression that the wall of the cyst was fused with the parietal pericardium. Our artist's interpretation of anatomic relationships is shown in Figure 7. The wound was closed without drainage.

The patient was placed in an oxygen tent. Convalescence was uneventful and he was discharged from the hospital two weeks after operation.

Pathologic Examination.—The cyst wall showed squamous epithelium, sebaceous glands, sweat glands and hair follicles. In some of the sections endometrium was found and epithelium resembling that of the upper and lower gastro-intestinal tract. Smooth muscle, normal fat and lymph follicles were also found. There was no evidence of malignant change. *Pathologic Diagnosis:* Cystic teratoma. The appearance September, 1939, is shown in Figure 8. The patient has remained entirely well, four years after operation.

A thorough search of the literature on this subject has not been made. In 1933, Hedblom[1] reviewed the literature on intrathoracic dermoid cysts and teratomata, and referred to three instances of intrapericardial lesions. One additional case was found since this report was made. King[2] refers to a report by Somolinos[3] which I have not obtained.

SYNOPSIS OF REPORTS OF FOUR INSTANCES OF INTRAPERICARDIAL LESIONS (*From the Literature*)

Reported by Joël[4]: This patient was a boy, age 14. There was no clinical history: Necropsy examination showed a tumor, the size of a hen's egg, inside the pericardial cavity. The tumor had a smooth, glistening surface. It was attached to the pulmonary artery, aorta and the heart. It was densely adherent to the pulmonary artery and penetrated the wall of the artery into its lumen. The mass overlay the left coronary artery and the left atrium, and contained gelatinous material, fat, lymph nodes, smooth muscle, cartilage and calcium deposits. It was classified as a teratoma.

Reported by Mouat[5]: This patient was a male, age 17. He was well until one year before death. The patient developed a mild fever, a cough and later on edema and ascites. The clinical diagnosis was tuberculous nodes in the mediastinum obstructing the superior and inferior venae cavae.

Necropsy examination showed an infected dermoid tumor in the mediastinum. The pericardial sac was greatly enlarged, but when this sac was opened it contained a large amount of pus from

FIG. 7.—Artist's interpretation of relationship between teratoma, right ventricle, right auricle, aorta and vena cava.

FIG. 6. — Photograph of a teratoma removed from within the pericardium.

which streptococci were grown. It also contained yellow fatty masses and long hair. The heart was embedded in the thick posterior wall of the cyst, and there was no sign of any pericardial sac. The cyst appeared either to have grown from the pericardium or in its growth to have become very completely adherent to it.

Reported by Grimm[6]: The patient was a child, age three months. There was no clinical history except a provisional diagnosis of idiopathic hypertrophy of the heart. Roentgenograms showed an enormous shadow in the mediastinum. Necropsy examination showed a broad fluctuating tumor which filled the anterior aspect of the chest, both lungs being pushed posteriorly. The mass had a thin membrane as a wall, and appeared milky in color. Inside the mass a light-colored lipoid fluid was found. The heart was inside the mass and was of normal size, shape and position. There was an

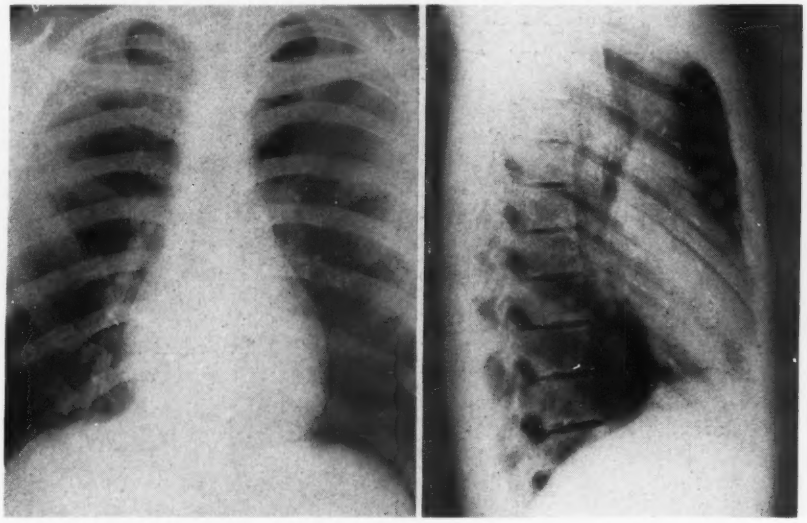


FIG. 8.—Appearance one year after operation. Note the silver clips in the lateral view. The cardiac silhouette is normal.

intimate association between the adventitia of the great vessels and the mass. The tumor was lobulated with small orange-sized growths of semicystic consistency. There were numerous pea-sized cystic compartments. On section, these were found to be multiloculated cysts with islets of homogenous tissue. In some of the cysts there was a semisolid material that looked like parboiled tapioca. In others a brain-like substance was found. Bacteriologic study showed no growth. Microscopic examination showed glial cells, choroid plexus, skin, sebaceous glands, sweat glands, muscle, mucous glands, cartilage, intestinal tract and hair follicles. The pericardium was milk-like in color but smooth and glistening. No evidence of malignancy. The author reported this case as one of intrapericardial teratoma. The relationship between the pericardium and wall of the cyst is not clear from the description.

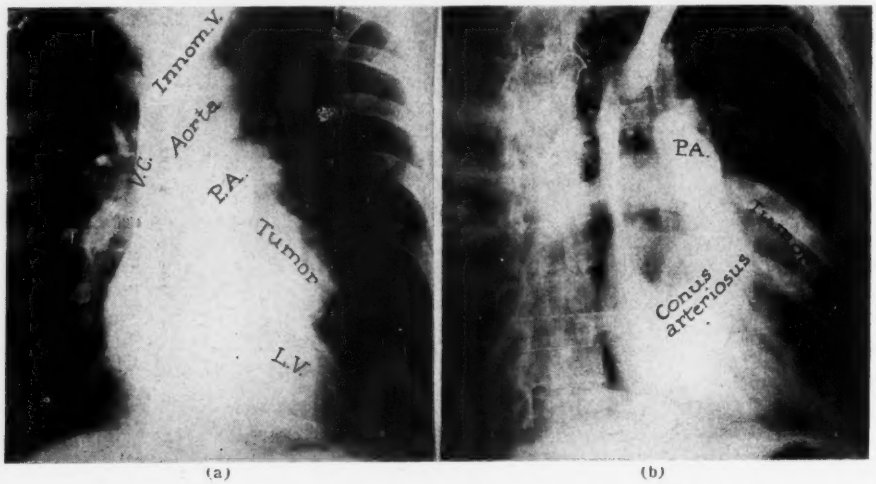
Reported by Jellen and Fisher[7]: The patient was a white female, age three weeks. Cough and dyspnea were present since birth. When six days old the child had convulsions. During the second week attacks of cyanosis appeared. A presumptive diagnosis of idiopathic hypertrophy of the heart was made. An aspirating needle inserted into the mediastinal mass yielded nothing, and a diagnosis of tumor was made. Necropsy examination showed a mediastinal mass within the pericardial sac. The pericardium over the mass was gray and thickened. When the pericardium was opened a tumor mass, 5 cm. in diameter, was exposed. It was firmly adherent to parietal pericardium and to the wall of the right ventricle, to the right auricle, to the great vessels and the transverse aorta. It overlay the bifurcation of the trachea. The pericardial sac contained 20 cc. of clear yellowish fluid. The heart was not enlarged and was hanging from the mass as a small appendage. The mass was a multilocular cyst. Microscopic examination showed various types of epithelium, choroid plexus, smooth muscle, cartilage and glandular structures resembling pancreas. There was no evidence of malignancy.

DISCUSSION.—It would appear that dermoid and teratomatous lesions situated within the parietal pericardium are of rare occurrence. The relationship between parietal pericardium and the wall of the cyst was not clear in any of the cases reported. It would appear that the parietal pericardium

may become an undistinguishable part of the wall of the cyst. These lesions can produce acute or chronic compression of the heart. They may become infected. Operative removal of the lesion was successfully accomplished in the case herewith reported. It would appear that operative removal is the only method of treatment.

TUMOR OF THE HEART, LEFT VENTRICULAR WALL

This patient was a white male, age 39, who complained of shortness of breath brought on by exertion, and a sense of constriction across the left side of the chest. He had been well until six years ago when these symptoms appeared. At first, the



(a) (b)

FIG. 9.—Roentgenograms of the heart. Diodrast was injected into a vein at the left elbow. (a) The innominate vein, the vena cava, and the pulmonary artery are shown. The tumor does not appear to communicate with any of the cardiac chambers. It is not an aneurysm. Calcification of the wall is present. (b) The right side of the heart wall is well visualized.

symptoms were mild but they increased in severity so that the patient had difficulty going to his office. These symptoms disappeared after he rested and they never came on while he was at rest, and suggested sclerosis of the coronary arteries as the probable cause.

The past history contained little of note. He had had typhoid fever from which he narrowly escaped death. There was no history of rheumatic fever, tuberculosis, diabetes or asthma. He had been quite active athletically as a youth, without any discomfort or any symptoms. The general physical examination, except the heart, was essentially negative. The skin over the face and neck showed scars of severe acne. An occasional extrasystole was noted. There were no cardiac murmurs. The arterial pressure was 120/72 mm. mercury. The electrocardiogram showed slurring of the QRS complexes and slight left axis deviation. There were no additional changes brought out by exercise. Doctor Feil carries out fluoroscopic examination of the heart as a part of his routine examination. In this patient fluoroscopic examination led to the correct diagnosis. Special studies with diodrast were undertaken by Dr. Eugene Freedman. Figure 9 shows the tumor in the heart. The left innominate vein, the vena cava, the right atrium, the right ventricle, the conus arteriosus, the pulmonary artery and the aorta can be identified. The tumor had a calcified wall. It did not fill with diodrast, and the pulsations of the lesion, as indicated by the kymograph film, were transmitted and were not expansile. The lesion, therefore, was not an aneurysm. I believed that we were dealing with a benign lesion of the heart—a tumor of some kind,

perhaps a dermoid. I also believed that this lesion produced the symptoms and, even though there was no precedent in the literature to support my attitude, I advocated operation.

Operation.—September 7, 1940: The third and fourth left costal cartilages were removed. The pericardial cavity contained a somewhat increased quantity of clear, slightly yellow fluid. The lesion in the heart was found. It was covered by epicardium. It could just be seen in our exposure (Fig. 10). Most of it was embedded in the myocardium but it did produce a bulge of about one or two centimeters from the general contour of the ventricle. An aspirating needle was introduced into it but nothing could be obtained. Two sutures were placed in the wall for traction and rotation. Considerable subepicardial fat was found around the base of the mass. This was dissected from the wall. The descending ramus and the circumflex ramus of the left coronary artery were in this fat. A branch from the descending ramus penetrated the wall of the lesion. This artery was cut and ligated. I had the impression that these arteries were displaced by the lesion. Sharp dissection was used to separate the wall of the tumor from the heart muscle. The two tissues were sealed intimately together and blunt dissection, most probably, would have torn the myocardium. (It is sur-

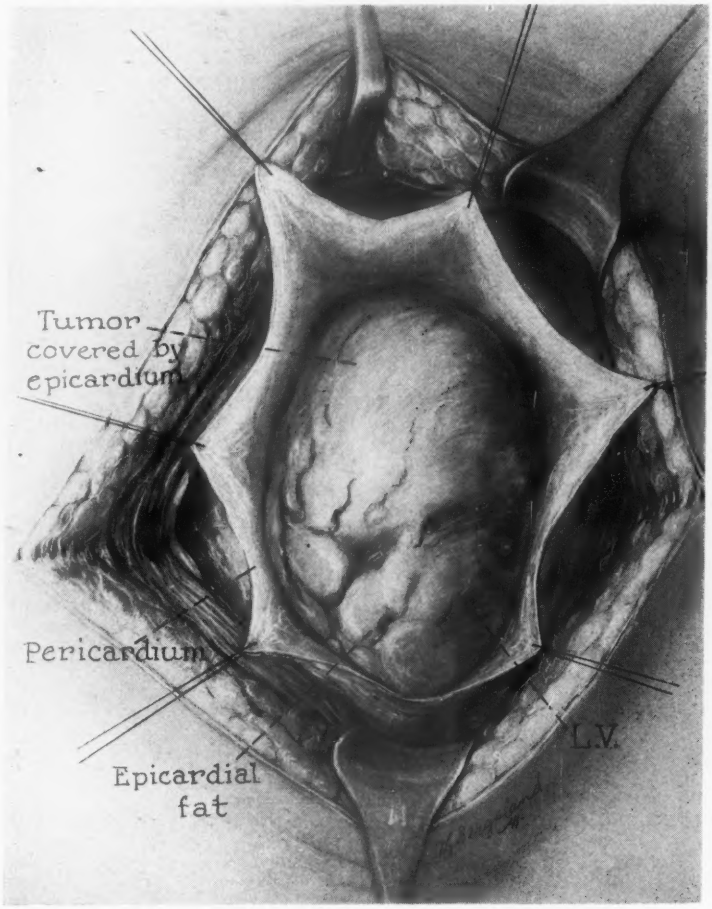


FIG. 10.—Appearance of the tumor after opening of the parietal pericardium.

prisingly easy to tear through the myocardium into the cavity of the heart). Sharp dissection was continued to the place shown in Figure 11. It appeared to be too much of a risk to continue further without knowing the nature of the tissues between the tumor and the cavity of the ventricle. The lesion was opened. The wall was calcified and was from one to three centimeters in thickness. The content was an amorphous material, homogeneous, with a consistency of packed clay. It was dark brown, and weighed 140 grams. This material was removed by currette and by washing with sodium chloride solution. There was no connection with the cavity of the heart nor could a week area be found by palpation. The calcified wall was dissected from the

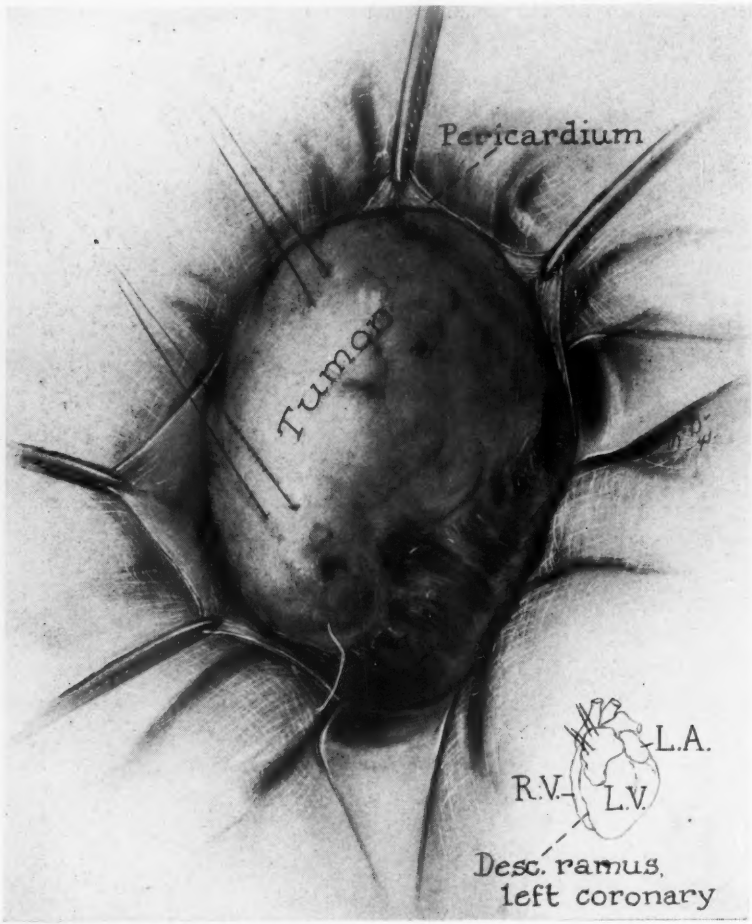


FIG. 11.—Subepicardial tumor of the left ventricle lying between the two branches of the left coronary artery. Traction sutures hold the tumor away from the myocardium. Sharp dissection was used up to this stage of the operation.

myocardium by sharp dissection. It was completely removed (Fig. 12). The bed consisted of cyanotic muscle. Digital palpation of the muscle was not carried out to determine how thick or how strong it was. The parietal pericardium was brought together by sutures. The wound was closed without drainage.

The patient made a satisfactory postoperative recovery. The electrocardiogram taken immediately after operation showed the T-wave in lead I to be inverted and

S-T in leads 1, 2, and 3 to be slightly elevated. Six days after operation, ventricular extrasystoles were noted. These disappeared two days later. The pulse rate ranged from 100 to 130 for several days after operation. Over a period of one month it came down to about 90. The patient was discharged October 7, 1940. He was worried about his condition after he left the hospital. He frequently counted his pulse. He became stronger and found that he could make the trip to his office without shortness of breath. The feeling of constriction across the left chest disappeared. The electrocardiograms became entirely normal. The patient appears to be cured.

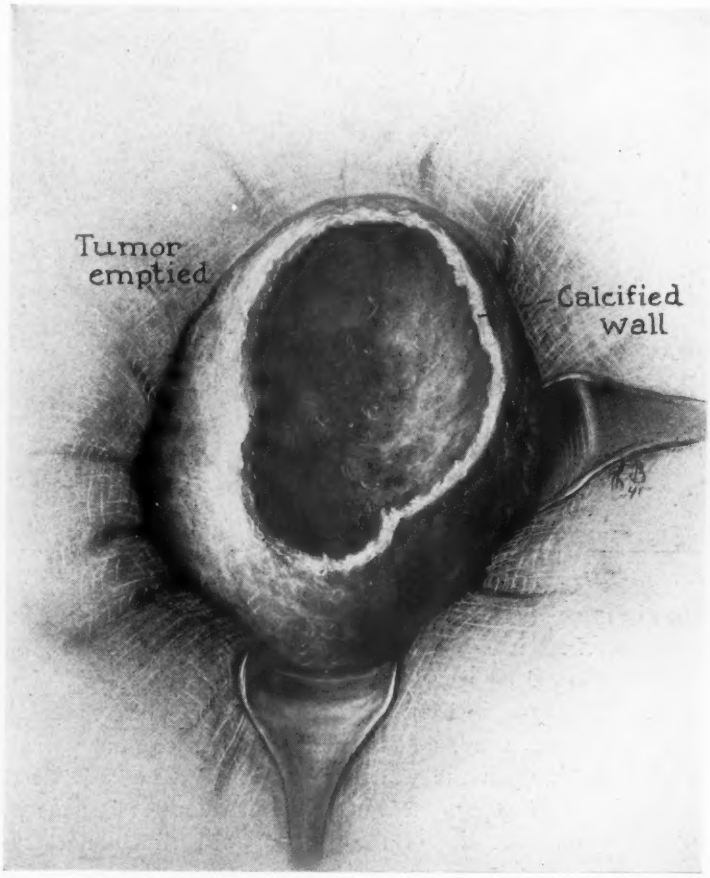


FIG. 12.—The wall of the tumor is incised. It was calcified. The contents were removed by curette. Complete dissection was then carried out successfully.

Pathologic Examination.—Dr. Howard Karsner: "The wall of the cyst shows nothing indicative of true neoplasm. It is simply a capsule of connective tissue, the interior aspect of which is necrotic and calcified. Chemical examination of the cheesy content of the cyst showed no iron. The lipid content determined on a specimen dried to constant weight and weighing 4.41 grams was as follows: Total fat 13.25 per cent; cholesterol 6.45 per cent. It was estimated that the same quantity of dried whole blood would contain total fat 3.59 per cent; cholesterol 0.81 per cent. The chemical examination gives no specific information because as far as can be learned there is nothing known of the composition of a hematoma after it has been present a long time."

COMMENT.—Before operation I thought this tumor might be a dermoid,

but we found no hair or other recognizable tissue in its contents. It had a calcified wall which did not show any epithelium or foam cells. It was not a xanthoma. The contents of the lesion had the consistency of packed clay, and between the gloved fingers it felt greasy. We cannot classify the lesion except to say that it does not appear to be a true neoplasm.

The question comes up as to whether an aneurysm will develop at the site of this lesion. I have carried out experiments for the purpose of producing aneurysms of the left ventricle. In these experiments a sphere of metal was introduced into the left ventricle by way of the auricle. The metal ball

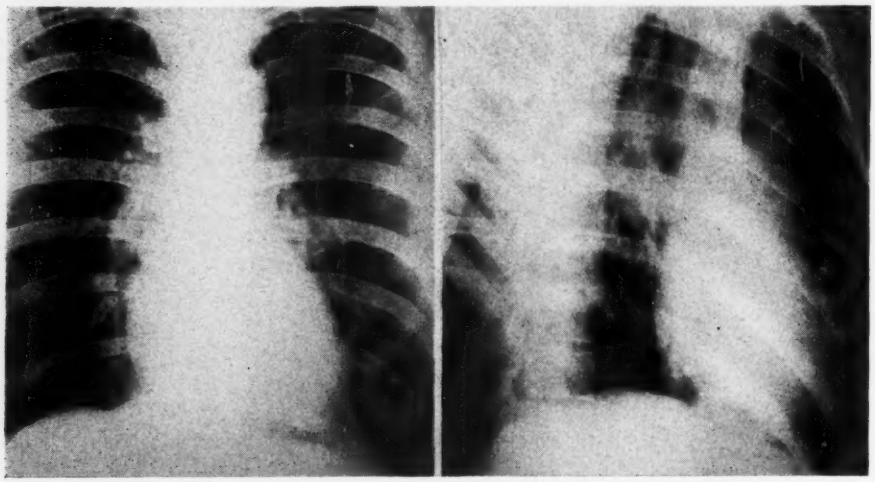


FIG. 13.—These roentgenograms appear to be normal, 18 months after operation.

was held firmly in position and pressure was applied to it through the myocardium. The myocardium could be crushed in this way between finger and metal. The metal could be seen through endocardium and epicardium. An aneurysm did not develop in these experiments. In other experiments the myocardium was sliced away, and in these experiments the parietal pericardium grafted itself upon the wound and an aneurysm did not develop. In this patient I should expect the parietal pericardium to become adherent to the myocardium at the site of the lesion and I should not expect an aneurysm to develop. The appearance 18 months after operation is shown in Figure 13. The patient has been completely relieved of his symptoms and appears to be cured.

I considered placing a free graft of parietal pericardium over the tumor bed, as illustrated in Figure 14, but felt that the parietal pericardium would become sealed to the tumor bed without placing such a graft. Recently I applied this method in the treatment of an aneurysm of the left ventricle.

DISCUSSION ON TUMORS OF THE HEART

A considerable literature exists on tumors of the heart. Most of these tumors are malignant and offer little or no possibility for surgical removal.

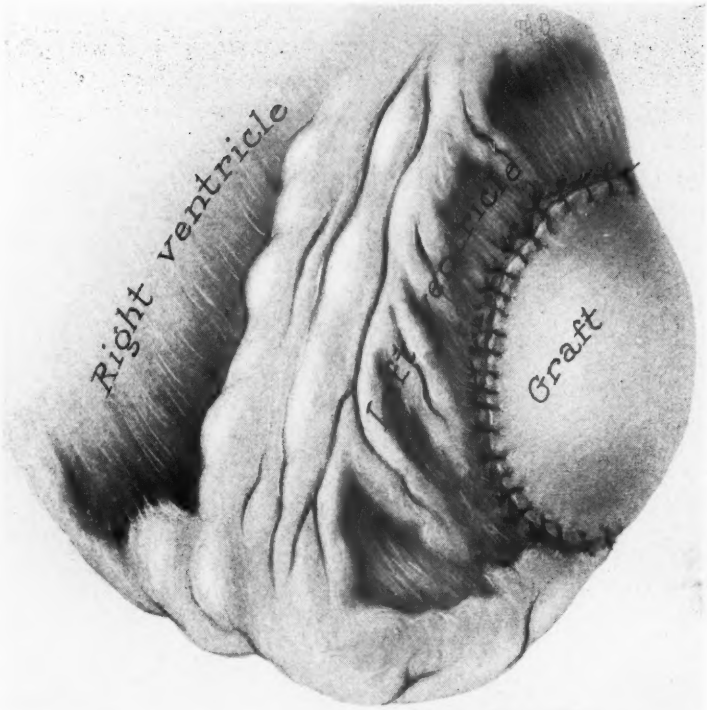


FIG. 14.—A free graft of parietal pericardium might be applied to the heart for the purpose of reinforcing a weakened area, such as might occur after the removal of a tumor or after myocardium has been severely bruised. This procedure might prevent rupture of a ventricle or the formation of an aneurysm of the heart.

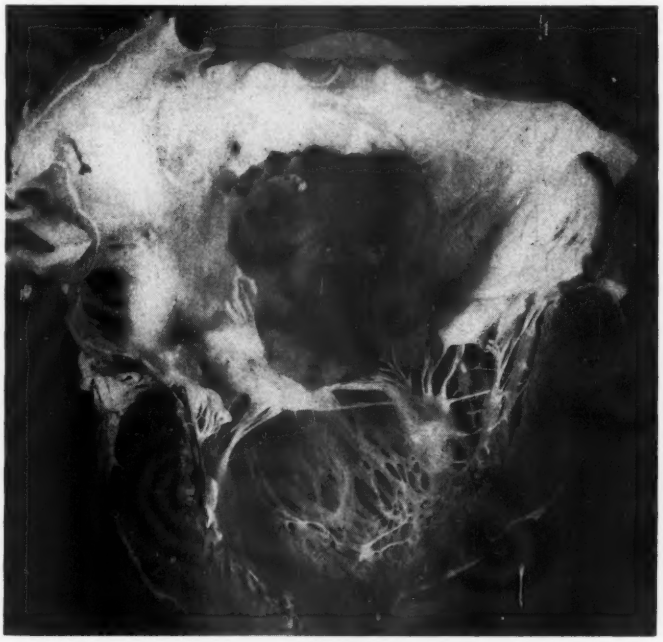


FIG. 15.—Photograph of the heart showing the tumor hanging through the mitral orifice. The tumor was attached by a slender pedicle. (Courtesy of Drs. Dexter and Work.)

Some of the malignant tumors possess pedicles that act as ball-valves in the mitral and tricuspid areas.[8] Even if such obstructive lesions could be removed the tumor would continue to grow. However, there are some benign tumors of the heart that seem to offer possibilities of surgical removal. I refer to the fibromata or myxomata that arise from the lining of the auricles and occlude the auriculoventricular orifices. One of these is illustrated in Figure 15, taken from an article by Dexter and Work.[9] This tumor was a

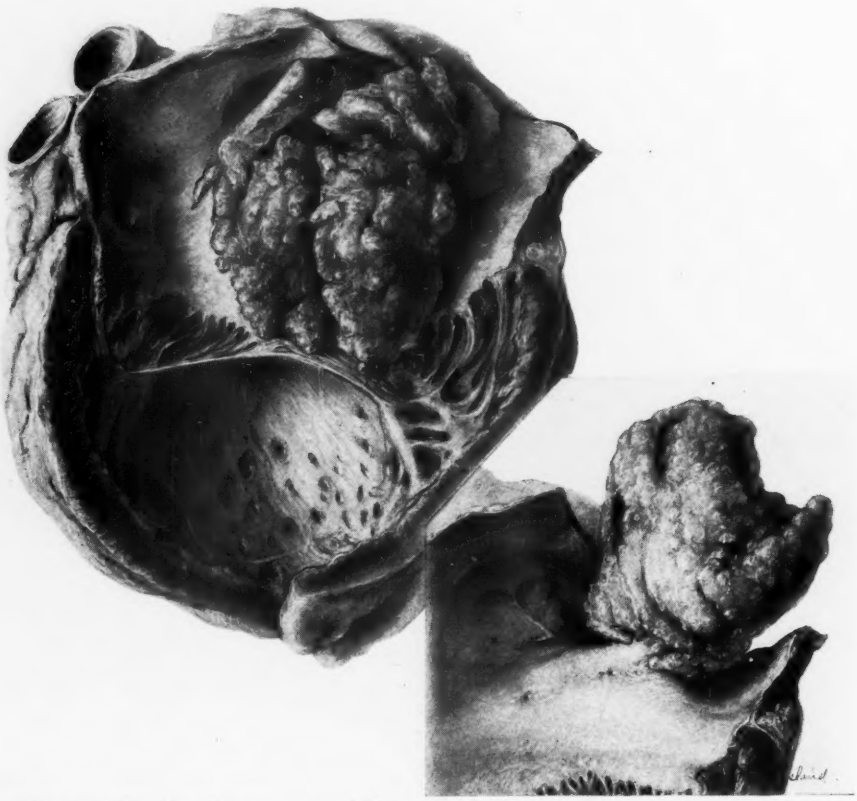


FIG. 16.—Drawing of myxoma attached by small pedicle to lining of left auricle. The inset shows the tumor lifted from the atrioventricular orifice. The pedicle was one centimeter in diameter. Removal of this tumor by operation seems to be within the realm of possibility. (Courtesy Institute of Pathology, Western Reserve University, Cleveland, Ohio.)

myxoma and originated from the subendothelial connective tissue of the endocardium of the left side of the atrial septum and had prolapsed into the orifice of the mitral valve, where, doubtless, it acted as a ball-valve, preventing the free flow of blood from the left atrium into the ventricle. The tumor was a pear-shaped lobulated mass which measured 4.5 by 4.2 by 3.5 cm. The tumor was attached by a pedicle and the small end of the tumor hung two centimeters below the valve ring. The tumor was soft and gelatinous. Another myxoma of the heart is illustrated in Figure 16. The left auricle was completely filled with an irregular nodular mass which protruded into the

atrioventricular opening. The mass was soft and jelly-like. It was attached to the auricular wall by a pedicle one centimeter in diameter. Weakness appeared two years before death. The left auricle became dilated. Circulatory failure developed. The clinical diagnosis was rheumatic heart disease and mitral stenosis. This patient showed embolic phenomena, and infarcts of spleen, kidneys, lungs and brain were found. These two cases appeared in Cleveland within a period of five years. Perhaps they are not so rare. A fibroma of the heart was reported by Houck and Bennett.[10] This tumor obstructed the mitral orifice into which it protruded and out of which it could be displaced. It was attached to the septum by a pedicle five millimeters in diameter. It was edematous, soft and jelly-like. It was not invasive. The tumor measured 4.5 x 4 x 3 cm. Strouse[11] reported a somewhat similar tumor in the right auricle; a myxoma, attached to the septum, and measuring 8 x 10 x 10 cm. This patient was intermittently well and ill, as though the tumor intermittently obstructed the circulation. The tumor was soft, jelly-like, and was not invasive. Another similar myxoma of the left auricle, attached to the septum, measuring 3.5 x 4 cm., was reported by Bennett, Konigsberg and Dublin.[12] It had a rubbery consistency, was relatively avascular, and was not malignant.

No doubt other similar benign tumors of the heart could be found in the literature. It would appear that the correct clinical diagnosis of a benign lesion of the heart, with the exception of my case, has not yet been made during life, and none of them have been operated upon. It seems to me that these soft, jelly-like, benign lesions arising from the endocardium could be removed by operation. It might be possible to remove them in much the same way as the neurosurgeon removes a soft glioma of the brain by means of strong suction. It does not appear to be impossible to insert a glass suction tube into the auricle for this purpose. It might even be possible to open the auricle and take out the tumor. We know that the circulation can be stopped for a period of three to five minutes without inflicting permanent damage to the brain. The great veins to the heart might be occluded for a few minutes. It is hoped that the method developed by John Gibbon,[13] of Philadelphia, might be applied to these surgical problems. Gibbon's method consists of removing venous blood, oxygenating the blood and delivering it to the brain under pressure. The heart has been taken out of the circulation for as long as 42 minutes in the cat, with recovery. This method seems to have great promise for the future.

While no other benign lesion of the heart has been recognized clinically or operated upon, it should be mentioned that Shelburne[14] took one step toward the surgical treatment of a malignant tumor of the heart. A correct clinical diagnosis was made by Shelburne, and this appears to be the first time that this has been done for a primary malignant lesion of the heart. Hemorrhage into the pericardial cavity took place in this patient and the blood was evacuated by operation. None of the tumor was removed.

SUMMARY

A case of intrapericardial teratoma is reported. There are only a few cases reported in the literature. This appears to be the first and only case in which the lesion was removed by operation. The lesion was completely removed, and the patient has been cured.

A case of a tumor-like mass located in the wall of the left ventricle is reported. The identity of the lesion has not been established. It was probably not a true neoplasm. This appears to be the first time that a benign lesion of the heart has been recognized clinically, and the only case in which the removal of the lesion has been carried out. The patient has had a good result, 18 months after operation. An aneurysm of the left ventricle has not developed. Benign tumors of the heart were discussed in relation to the problem of removal. A method of grafting a segment of pericardium or fascia lata upon the ventricle to reinforce the weakened area and to prevent the formation of an anuerysm is illustrated.

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POSTOPERATIVE THROMBO-EMBOLIZATION

The Platelet Count and the Prothrombin Time After Surgical Operations: A Simple Method for Detecting Reductions and Elevations of the Prothrombin Concentration (or Activity) of the Blood Plasma.

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IT HAS BEEN DEMONSTRATED that postoperative thrombosis and embolism may be prevented by the use of the anticoagulant heparin.[1, 2] Because this substance possesses certain objectionable features, such as its cost, the necessity for continued intravenous administration, and adverse reactions in some patients, it was considered possible that other anticoagulants might, under certain conditions, be more preferable.

In a recent series of publications, K. P. Link, and his associates,[3, 4, 5, 6, 7] of the Wisconsin Experiment Station, revealed an outstanding work in which they isolated, identified, and synthesized the causative agent of the hemorrhagic "Sweet Clover Disease of Cattle."[8, 9, 10, 11] This substance –3, 3′ methylenebis (4-hydroxycoumarin)—in adequate dosage decreases the coagulability of the blood *in vivo* by reducing its prothrombin level (or activity). Before applying this material as a possible prophylactic agent against postoperative thrombo-emboli it was desirable to investigate, first, the coagulability of the blood and particularly the prothrombin times during the postoperative periods.

Changes in the clotting mechanism of the blood, as expressed by alterations in the platelet count, after surgical operations and parturition, have been demonstrated by Hueck,[12] and Dawbarn, Earlam and Evans.[13] They showed that there is initially a temporary reduction in thrombocytes, followed on or about the sixth day by a more or less sharp rise, after which the count gradually recedes to normal on the tenth to the fourteenth day. It was emphasized that most of the fatal cases of thrombo-embolization occurred during this interval of thrombocytosis.

We have repeated these studies in 23 patients after surgery, and, in addition, have studied the prothrombin time of the plasma.

Complete blood counts were made at least every third day. The platelet count and coagulation time and the plasma prothrombin time were estimated daily, except Sunday.

The technic for estimation of the prothrombin time was that described by Link, and his students, (based on the single-stage method of Quick[16]), ex-

cept that Russell snake-viper venom[14, 15] was substituted for the thromboplastin-calcium chloride mixture, as prepared by Link, and his coworkers.[4*]

Briefly stated, the method is as follows. Four point five cubic centimeters of freshly drawn venous blood are added to 0.5 cc. M/10 sodium oxalate. Clear plasma is obtained by centrifuging. One-tenth cubic centimeter of plasma is added to 0.1 cc. of venom and placed in a constant temperature bath for five minutes. To this is quickly added 0.1 cc. M/40 calcium chloride, which has been kept at the same temperature, and the time elapsing before fibrin clot formation is noted with a stop-watch. The same procedure is then repeated with 0.1 cc. of 25 per cent plasma in place of whole plasma.

Dilution of one part of whole plasma with three parts normal salt solution permits of a clearly detectable and satisfactorily reproducible end-point. It has been found that, in man, higher dilutions, especially when the prothrombin time is considerably prolonged, often do not.**

It has been found, with this method and using venom, that the average difference between the prothrombin time of whole and of 25 per cent plasma obtained from apparently normal individuals is about ten seconds.† It has been found also, that prolongation of this difference occurs when the prothrombin level (or activity) of the blood becomes reduced below normal, and that shortening of this difference takes place as the concentration (or activity) of prothrombin in the blood increases. A state of hyperprothrombinemia is, theoretically, a possibility. It appears that a lessening of the difference, as revealed in this method, below that observed in normal plasmas might indicate such a condition.[17]

This report embraces the above detailed studies on 23 postoperative patients.

DATA

Results. On the first to about the fourth day after surgical operations the platelet count was relatively decreased in eight of 23 cases. Commencing about the fifth or sixth day, and as late as the fourteenth day postoperative, an increase in the circulating thrombocytes was observed in 15 cases, varying in degree from about one and one-half to about twice the count prior to the time this elevation commenced. The duration of this platelet rise was variable, the shortest being two days and the longest a fortnight. The coagulation time, as estimated by the three-tube method of Lee and White,[18] showed little deviation from the normal (Chart 1).

* In the present series venom was used exclusively. In a subsequent study the thromboplastin-calcium chloride mixture of Link, *et al.*,[4] is being used.

** This applies when venom is used. It has been found that 12.5 per cent plasma is better suited for this estimation when Link's thromboplastin-calcium chloride mixture is used.

† Each laboratory should determine this value by the particular method used. By the technic described, the arithmetic mean has been found to be 10.5 sec., and the standard deviation s.d.±2.4, the minimum 7, and the maximum 16.5.

In four of the postoperative patients who showed a fall in the platelet count after operation (and seven others who did not), there was also observed, concomitantly, an extension beyond that seen in normal plasma of the difference between the prothrombin time of whole and of 25 per cent plasma. This latter was due, in most part, to a greater prolongation of the 25 per cent plasma prothrombin time in relation to that of the whole plasma. When the

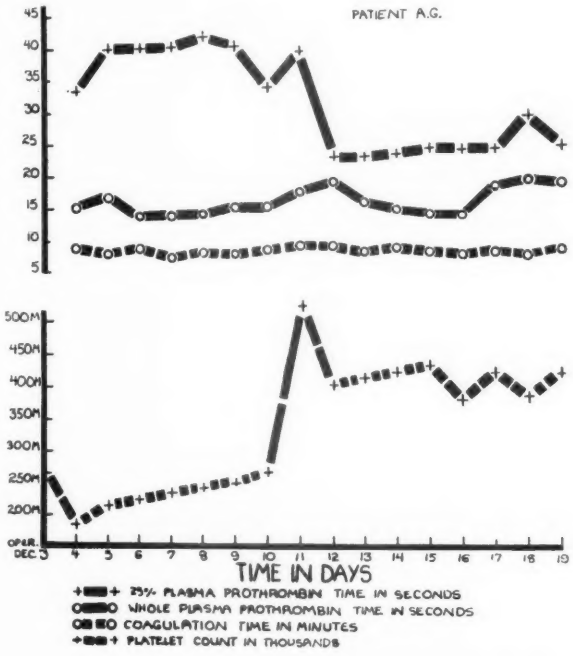

CHART 1.—The platelet count, coagulation time and prothrombin times of a case, illustrating the initial fall in platelet count and its subsequent rise on the seventh (7th) day after operation. The coagulation time (Lee and White method) changed slightly. The widened initial difference in prothrombin time between whole and 25 per cent is shortened simultaneously with the thrombocytosis.

circulating thrombocytes increased, this difference shortened, due, chiefly, to a reduction in the 25 per cent plasma prothrombin time. This was noted in 14 cases, in three of which, however, no significant alteration in the platelet count occurred. In three cases, in which the difference fell to below three seconds on two successive days, thrombophlebitis was found to be present, and in two of these, pulmonary infarction occurred. No change in the blood counts was observed which could be correlated with the alterations in the platelets and prothrombin time described above (Chart 2).

DISCUSSION.—The precise mechanism responsible for the initial fall in the platelet count following surgical operations is not clear. Anesthesia is not the cause, for we have detected it after local as well as general narcosis. Others have made the same observations.[13] It has been suggested that an

TABLE I

STATISTICAL DATA OBTAINED IN 23 POSTOPERATIVE PATIENTS

				Platelets (in thousands)				Difference between Whole and 25% Plasma Prothrombin Time (in seconds)				
Number	Patient	Sex	Age	Initial Fall.*	Day Post-oper.	Rise*	Day Post-oper.	Initial Diff.*	Day Post-oper.	Diff.* Reduced to	Day Post-oper.	Operation
1	A. G.	M.	27	260 to 190	1	260 to 510	7	19	1	4	7	Herniotomy
2	B. T.	F.	32			220 to 360	5	40	1	15	5	Appendicectomy
3	B. A.	F.	52					17	1	5	3	Pelvic repair
4	L. P.	F.	40			240 to 420	4	20	1	6	4	Appendicectomy
5	H. B.	M.	30			220 to 330	5	15	1	5	5	Appendicectomy
6	A. B.	M.	39			400 to 560	3	15	1	2	3	Amputation, gangrene
7	J. P.	M.	41			190 to 360	4	9	1	4	4	Hemorrhoidectomy
8	M. D.	M.	38			260 to 490	8	10	1	8	4	Appendicectomy
9	O. A.	M.	72	493 to 305	3	300 to 400	6	30	3	10	6	Revision of stump
10	H. C.	M.	29	300 to 240	3	220 to 340	7	21	3	4.5	6	Herniotomy
11	S. G.	M.	32			208 to 360	12-14			2	14	Herniotomy, first seen on 10th day postoper.
12	J. G.	M.	22	230 to 150	5	180 to 367	10	10	1			Open reduction—Kirschner Wire
13	G. L.	M.	27			220 to 290	4	9	1	4	4	Hydrocele
14	M. E.	F.	16			240 to 300	7	8	1	2	2	Appendicectomy—103° fever on 2nd day postoper. only
15	A. R.	F.	50			260 to 360	2	25	1	13	2	Appendicectomy
16	H. G.	M.	29					20	2	5	7	Herniotomy
17	F. D.	M.	27			260 to 390	4	7	2	8	4	Open reduction—Kirschner Wire
18	H. F.	F.	36							2	14	Pelvic repair. Thromboemboli first seen on 10th day postoper.
19	M. S.	F.	40					8	2			Appendicectomy
20	A. K.	M.	42	225 to 170	2							Appendicectomy
21	M. K.	M.	49	309 to 215	3							Gastrostomy
22	C. McC.	M.	27	300 to 250	2							Hemorrhoidectomy
23	P. R.	M.	30	419 to 202	2			16.5	2			Skin graft

* Blank squares indicate no change.

agent is liberated which depresses the thrombocytes along with other constituents of the blood, including the proteins.[12, 23] The concomitant prolongation of the prothrombin time suggests similar inhibitory effects upon the formation or activity of prothrombin. This hypoprothrombinemia may be controlled by vitamin K administration. None of the patients of the present series had received vitamin K.

The explanation for the subsequent thrombocyte and prothrombin increase is lacking. It is important to point out the parallelism that appears to exist between these two coagulation bodies—platelets (thromboplastin) and prothrombin. Whether other related factors, such as fibrinogen, similarly increase is now being investigated.

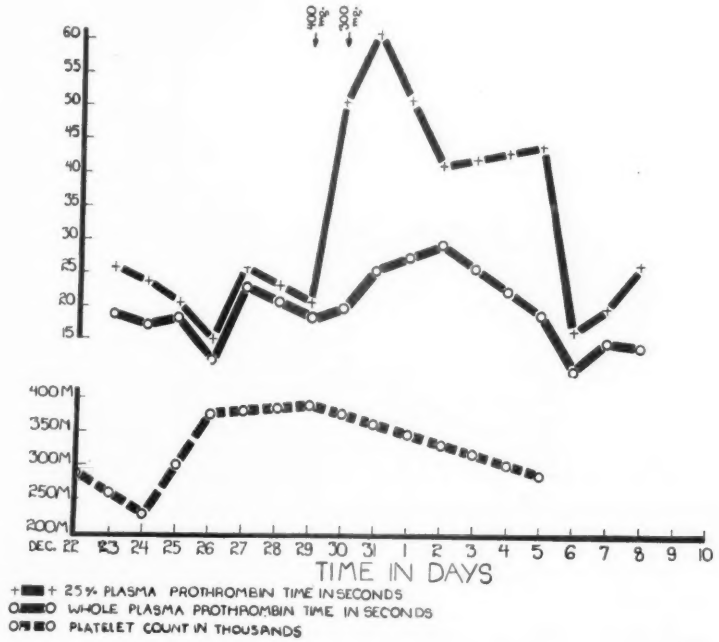

CHART 2.—Illustrates marked shortening between whole and 25 per cent plasma prothrombin times immediately preceding pulmonary infarction which occurred on December 27. The dosages noted—400 and 300 mg. on December 28, and 29, respectively, are of 3, 3′ methylenebis (4-hydroxycoumarin) given orally. Note effect upon the prothrombin level (or activity).

Our findings, and those of previous workers,[12, 13] correspond in respect to the thrombocytes. There is some lack of agreement between the coagulation times as observed by us and those of Dawbarn, Earlam, and Evans,[13] and this is to be explained by difference in method. They used a capillary-tube method, hence, their specimens of blood might have been contaminated by tissue juices, which are known to possess coagulating properties. In any event, as estimated by the method at hand, the whole blood coagulation was not appreciably altered beyond the limits of normalcy.

The difference in prothrombin time between that of whole and 25 per cent plasma appears to be significant. Normally, there is in the blood an excess of prothrombin beyond that needed for normal coagulation.[16] The extent of this excess seems variable in certain diseased states (liver), although under apparently normal conditions it is constant within the limits detectable by the method used here. The whole blood prothrombin time remaining within

normal limits and the difference increasing, the indication appears to be that the excess only is being depleted while the basic essential normal prothrombin level (or activity) is being maintained. However, should the whole plasma prothrombin time also show progressive prolongation it would signify that this plasma prothrombin content (or activity) was also being reduced and might be approaching a hazardous level.*

An inverse relationship between the difference in prothrombin time between that of whole and of 25 per cent plasma, and platelet count, is demonstrable in those postoperative cases in which alterations in the number of circulating thrombocytes was observed. In other words, the prothrombin concentration (or activity) and the platelet count increased *pari passu*. Prothrombin does not have its origin in the platelets,[24] nor is it necessarily influenced by thrombocytopenia occurring under other conditions. It appears that the tissue of origin of prothrombin (liver?) and of the platelets or the megakaryocytes are similarly affected after surgery.

The data indicate that these substances (prothrombin and thromboplastin) which take part in the process of coagulation of the blood increase in concentration and/or activity commencing about the sixth day after operation. This is the interval when the incidence of postoperative thrombo-embolization is greatest.[25] It is believed that there is casual relation between these two events. The finding that the concentration (or activity) of prothrombin is greatest (as shown by the shortest differences between the whole and the 25 per cent plasmas) in those instances in which thrombo-embolic phenomena had taken place, might also be interpreted as indicating a possible relationship between these events. Whether there actually occurs a hyperprothrombinemia remains to be further demonstrated. It is also not known whether the change in the blood vessels precedes the increased prothrombin level (or activity) or follows it. The mechanism of blood coagulation involves several reactions. Although these overlap the process of blood clotting is, nevertheless, continuous and progressive. Hence, inhibition at any one stage stops all the succeeding reactions. Thus, the indication for a therapy which is capable of limiting the coagulations of the blood in such a manner becomes apparent. The substance 3, 3′ methylenebis (4-hydroxycoumarin) prevents or inactivates prothrombin in man.[20, 21] If this can be administered with adequate safety, and so that the extent of its action can be predicted with some approximation, it might prove appropriate as a preventive against postoperative thrombo-embolization, especially in view of the finding that the prothrombin level (or activity) appears to be increased at the time that thrombosis occurs. It does not appear necessary to administer anticoagulant therapy to every patient after surgery as a routine measure, for the incidence of postoperative thrombo-embolization is about one per cent of major surgical cases.[24]

* This is especially important in following the effects of dicoumarin therapy.

The procedure being followed by us is as follows: Prothrombin estimations are made daily after surgery. In those instances in which the difference between whole and 25 per cent plasma prothrombin time becomes progressively shortened to below six seconds for two successive days, the dicoumarin compound is given. Its effect is observed by following the prothrombin times (Chart 3).

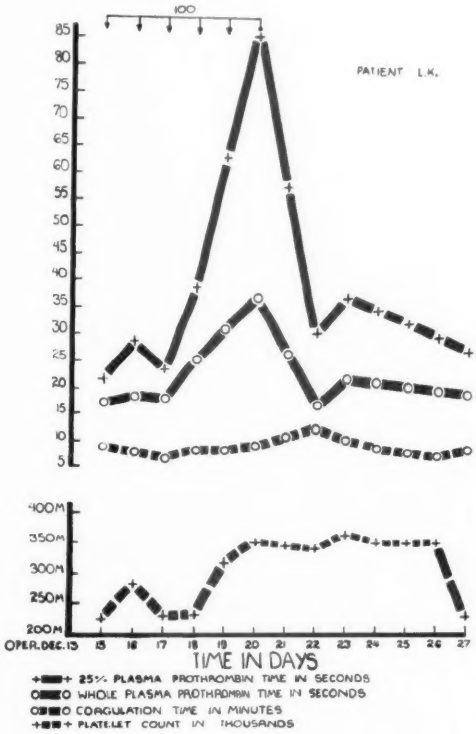

CHART 3.—Illustrating the effect of 100 mg. daily by mouth for six successive days commencing on the second day after operation of 3, 3′ methylenebis (4-hydroxycoumarin). There were no complications and no thrombophlebitis in this case.

CONCLUSIONS

Evidence is presented which confirms the findings of previous observers, [12, 13] that the platelet count may be (1) low during the first three to five days; and (2) elevated commencing about the sixth to tenth day, and continuing for from two to 14 days after surgery.

It has been found (similar to reported observations[19]) that the prothrombin concentration (or activity) may be low, initially, after operation.

In 14 of 23 cases the prothrombin level (or activity) was found to increase, usually concomitantly, with the thrombocytosis occurring on the sixth to tenth day following operation.

In three cases of postoperative thrombophlebitis (two of which also had pulmonary infarction), the prothrombin content (or activity) was highest of all the cases examined. The suggestion is offered that this increase in coagulating substances in the blood is related to the development of postoperative intravascular thrombotic phenomena.

Studies are being made to determine the safety and efficacy of 3, 3′ methylenebis (4-hydroxycoumarin) as (1) a prophylactic against postoperative thrombo-embolization; and (2) as a treatment of venous thrombosis and its complications.

The authors desire to express their thanks and appreciation to the following for suggestions and helpful advice in the prothrombin studies: Professor Karl Paul Link, Dr. Mark Arnold Stahmann, Dr. Harold A. Campbell, and Dr. Ralph Overman.

The dicoumarin compound was obtained through the courtesy of Dr. George R. Hazel, of the Abbott Laboratories, and Dr. K. K. Chen, of the Eli Lilly Co.

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THROMBOSIS AND GANGRENE OF RIGHT ARM, ASSOCIATED WITH POLYCYTHEMIA VERA: ITS RELATION TO "EFFORT THROMBOSIS"

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AN UNUSUAL CASE is herewith reported both because of its own particular interest and because it brings out the occasionally overlooked association of a general disease with a local vascular change. Despite its rarity the case has practical applications.

Case Report.—Hosp. No. 11707: E. J. F., white, male, age 76, was admitted, January 29, 1940, to the Chestnut Hill Hospital, complaining of numbness, tight stiffness, and aching in the right arm. The present illness began eight days before admission, when he noticed these sensations along the inner side of the arm but had no great discomfort or concern till four days later, when redness and swelling ensued, which extended above and below the elbow. A red streak could then be traced halfway to the shoulder. Hot wet dressings of magnesium sulphate solution were applied, without effect however.

The only other complaints were occasional precordial pain and some discomfort from an ulcer in a discolored area above the right ankle. There was also some discharge from an old scar on the right foot. He gave a history of having had typhoid fever and "intermittent fever" at age 26. A partial amputation of the right foot had been performed for an injury 43 years ago. His family and social history had no bearing on his present condition.

Physical Examination.—This disclosed a somewhat red-faced, elderly male, surprisingly well preserved for his age. Temperature 98° F., pulse 94, respirations 24. Blood pressure 108/96. His right arm was swollen and reddened from the shoulder to the hand. The pulse at the wrist was good. The arm did not feel hot. No enlarged axillary nodes were palpable. Further examination was negative, except that the legs exhibited brownish pigmented areas above each ankle, an ulcer being present on the right. The right foot had been partly amputated, and the stump had an open area in the scar. Pulses were not palpable in either leg below the popliteals. *Clinical Diagnosis:* Thrombosis or cellulitis of right arm; general arteriosclerosis, particularly marked in the legs.

Course.—On the day following admission a blood examination showed Hb. 160 per cent (26.5 mg.), R.B.C. 5,750,000, W.B.C. 10,500, P. 86 per cent, L. 8 per cent, M. 4 per cent, and E. 2 per cent. Repeated frequently, specimens being taken from various sites, the hemaglobin and red cell values were always well above normal, the highest hemaglobin value being 175 per cent (2-9-'40), and the highest red cell count 8,350,000 (2-12-'40). The only platelet count (3-6-'40), was normal (358,470).

Wet dressings of a saturated solution of magnesium sulphate were applied for the first two days, but the swelling progressed to the shoulder and neck (Fig. 1). The diagnosis of polycythemia vera being tentatively made, a medical consultation was asked. At this time the right arm was warm and the radial pulse full and bounding. There was no tenderness. Swelling was present, with pitting on pressure. Blood pressure was approximately the same in both arms. There was bulging of the supraclavicular fossa and the overlying skin was more dusky than on the opposite side. The sternal end of

the right clavicle appeared to be pushed forward. There was fullness and diminution in breath sounds in both the supraclavicular and suprascapular regions. A firm cord-like structure was felt and seen in the supraclavicular region extending up the neck (Fig. 2) which did not pulsate. No Horner's syndrome was noted.

The possibility of a sulcus tumor obstructing venous and lymphatic drainage was entertained, which raised the question of interference with pulmonary circulation as the cause of the change in blood count. A roentgenogram (1–31–'40) showed no tumor, aneurysm or sclerosis of pulmonary vessels.

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III

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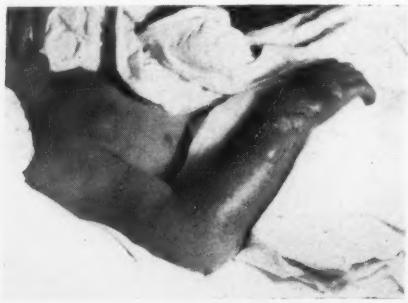

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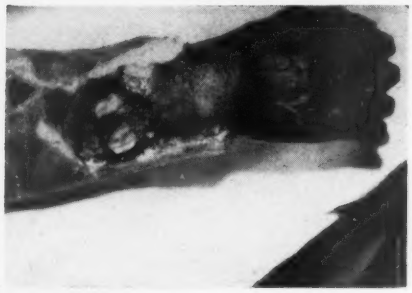


VI

Fig. 1.—Swelling of arm, shoulder and neck in early stage of condition.

Fig. 2.—"A firm cord-like structure is felt in the supraclavicular region extending up the neck" —the external jugular vein.

Fig. 3.—Early signs of wet gangrene in hand.

Fig. 4.—Progression of wet gangrene up forearm.

Fig. 5.—Full extent of gangrenous process.

Fig. 6.—Appearance immediately after operation.

The swelling progressed, the upper right quadrant of the chest wall and the right side of the neck being involved. Pulsations at the right wrist were still felt but evidence of early wet gangrene (Fig. 3) began to make their appearance in the hand. On 2–7–'40 phenylhydrazine hydrochloride was begun and continued one week. Novocain injections into the cervical sympathetic ganglia were given (2–7–'40 and 2–9–'40) in an attempt to combat vasospasm and promote vasodilatation in the collateral circulation, with little success. General supportive measures were, of course, being used continuously.

Sterile dressings and dry heat were applied to the right hand and arm, but the gangrene progressed steadily, first involving fingers then hand and forearm (Fig. 4). The initial edema and cyanosis of the chest wall, supraclavicular fossa and neck gradually lessened. On 2–14–'40, Dr. Hugh Montgomery felt the gangrene to be due to venous and lymphatic blockage. Oscillations recorded by him showed pulsation to be excellent in the uninvolved part of the right arm, demonstrating the freedom from involvement of the upper arterial tree. He suggested culturing of fluid from the blebs which was done and showed (1) Hemolytic and nonhemolytic *Staphylococcus aureus.* (2) Nonhemolytic *Staphylococcus albus.* (3) Nonhemolytic streptococcus (the organism usually found associated with lymphatic blockage). He also suggested reducing the blood count to normal by phenylhydrazine or bleeding. This drug was again ordered and given for two days, then discontinued. Bleeding was not done, localization of the gangrenous process already having begun, and swelling receding elsewhere.

With this localization of the gangrene, the patient began to improve generally, and from the surgical standpoint it was felt that amputation was inevitable but should wait upon the establishment of a clear line of demarcation.

Various laboratory procedures were carried out during the waiting period. Most of the results were within normal limits. Urine showed no erythrocytes or casts and was otherwise negative. Blood chlorides were low (391 mg. per cent on 2–13–'40) the only time taken. Clotting time (2–5–'40) was three minutes. Fragility test on the same day showed hemolysis beginning at 0.48 and complete at 0.36. Blood urea was 15 mg. per cent, blood sugar 100 mg. per cent, and the Wassermann and Kahn negative.

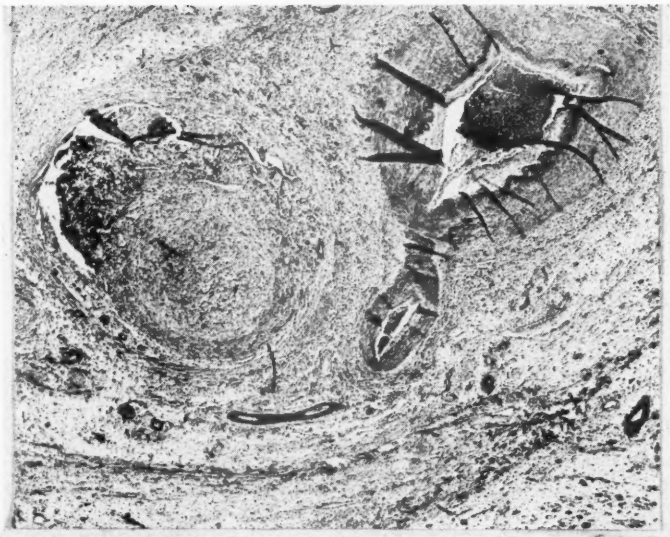
The gangrenous process had arrested itself at midforearm (Fig. 5) by 3–30–'40, 69 days after onset. On that day, with the patient under gas-ether anesthesia, an incision was made following the line of demarcation in the midforearm, going through all soft tissue down to the bones. By so doing a flexor surface flap was made. Bleeding was copious (just proximal to the line of demarcation) from both the radial and ulnar arteries, which were clamped and tied. Periosteum on both radius and ulna was raised and stripped upward and both bones were sawed through at the junction of the upper and middle thirds of the arm. The remaining bleeding vessels were ligated and the lower flap was turned up and sewn to the upper by a few through-and-through sutures of silkworm gut (Fig. 6). The patient stood the operation well and had a fairly smooth postoperative course. There was some infection of the stump, but of a nonvirulent and well localized type. It took about 12 weeks before healing was complete. Generally, the patient was definitely improved after the amputation. His blood count three days later showed Hb. 78 per cent, R.B.C. 3,910,000, where a week before it had been Hb. 113 per cent, R.B.C. 5,170,000. It remained at this lower level until discharged (4–29–'40).

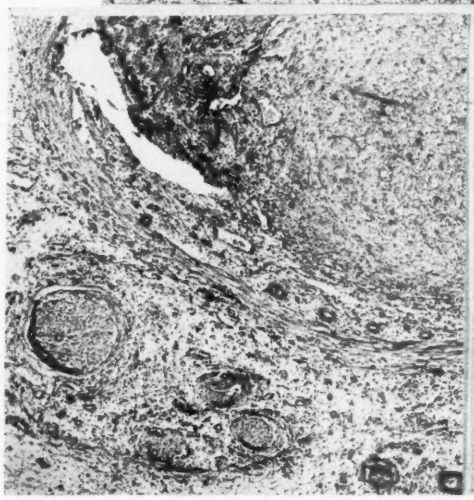
Examination of severed hand and lower arm showed general tissue necrosis and thrombosis of veins and arteries as well as some perivascular inflammatory changes.

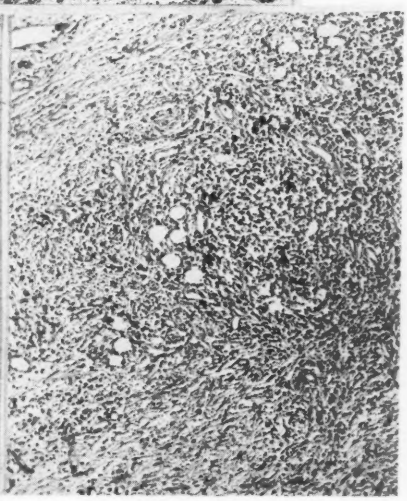
Following discharge the patient attended the Clinic, and his arm, as stated, gradually healed. No evidence of the recent involvement remained in the upper arm. The cord-like external jugular had returned to normal. The swelling and cyanosis in the shoulder and chest had disappeared. He did have some more trouble with his leg ulcers, however. Also, the stump of the right foot seemed more open, but in general he was in excellent condition.

In the course of a few more weeks the blood count again reached its former high level and he was readmitted to the hospital (8–2–'40). At this time his spleen was definitely enlarged and palpable. Sudden death occurred (8–31–'40). At autopsy, the following significant findings were noted: (1) Coronary thrombosis with myocardial infarction. (2) Bone marrow showing erythroblastic hyperplasia. (3) Splenic enlargement. (4) A well healed stump of the right forearm. *Pathologic Diagnosis:* 1. Polycythemia vera. 2. Coronary embolism.

VII







VIII IX

FIG. 7.—Radial artery and vein. Note almost complete organization of thrombus in vein while artery still shows some unorganized clot in its lumen. This points to the venous origin of the process and the retrograde course of the clotting.

FIG. 8.—Involvement of smaller veins in the complete thrombosis is seen. These are on the periphery of the radial vein and while probably not true vasa vasorum may have received the flow from those vessels. The small arterial channels are all partly open (loose clot may have been washed out).

FIG. 9.—Definite subacute and chronic inflammatory reaction in tissue adjacent to artery and vein. The apparently open lymph spaces are really fat cells whose contents have been dissolved. Few really open lymph spaces are visible.

Discussion.—Thrombosis is not at all unusual as a complication of polycythemia vera (Vaquez's or Osler's disease). In fact, about one-third to one-half of the deaths ascribed to this disease are traceable to thromboses of mesenteric, cerebral, coronary or other vessels. But the involvement of the veins of an upper extremity is extremely rare. Stover and Herrell,[1] of the Mayo Clinic, report one such case, which almost paralleled our own, and in reviewing the literature they found only one other case resembling theirs, that cited by F. Parkes Weber,[2] which was really a widespread thrombosis involving both arms and both legs. An article on the "Vascular Complications of Polycythemia", by Norman and Allen,[3] contains no reference to thromboses in the arm, nor does a more recent article by Zeiter.[4] There are, however, other references to thrombosis of vessels in the upper extremity in association with polycythemia vera in the European literature. Interest in the condition known as "effort thrombosis" began there with the first description of it by von Schrotter,[5] in 1884, and its continued study since has led to the finding of polycythemia in association with some of the cases. Lohr[6] reports six cases of thrombosis in the upper extremity. In two of these polycythemia was definitely present; in two more, its presence was suspected; in the last two, studies were too incomplete to establish it or rule it out. He cites the association of these two conditions in one case reported by Winterstein,[7] and in three cases by Julius Bauer.[8] The above cases plus the Mayo case and our own, account for eight definite instances where polycythemia and a thrombosis in the upper extremity occurred concomitantly. There are probably more. Our case has the questionable distinction of being the only one in which the process went on to gangrene formation, resulting in partial loss of the limb.

Thrombosis of the axillary or subclavian veins from other causes is not uncommon. J. R. Veal[9] covers this subject of thrombosis in the upper extremity very thoroughly, and reports 17 cases—none, however, in association with polycythemia vera. He divides his cases into: I. Primary—(A) Thrombophlebitis (bacterial); and (B) Phlebothrombosis (nonbacterial, traumatic or effort thrombosis). II. Secondary—(A) Thrombophlebitis from regional infection; and (B) thromboses from malignancies of the axilla and chest. We quote this logical classificatoin in order to indicate that our present case would fall under group I–B, and to point out its possible relationship to that other condition listed under the same heading "effort thrombosis"—a thrombosis accompanied by a fairly massive swelling of the involved part following some slight trauma, unusual effort or "inappropriate movement." We suggest the possibility, at least, of the presence of polycythemia, often unrecognized, as an underlying factor in some of these cases of "effort thrombosis," where one is often hard put to trace even the slightest abnormal effort as a precursor of the lesion. This suggestion is not original with us, the theory having been advanced very definitely by Lohr,[6] in 1934. But, his suggestion seems to have been overlooked by sub-

sequent writers on the subject. We hold his theory to be of prime importance and worthy of careful attention in the study of future cases of a condition so little understood that Matas[10] describes it as "a complex syndrome of polyvalent causation." Matas also gives a clue as to why polycythemia has not been found more often when he writes "*in the few cases in which careful hematologic studies have been made there are no notable blood changes———.*" Again, one must remember that the blood count and hemoglobin values vary from time to time in polycythemia, and if a routine count be taken at a time when these values are at or near normal, polycythemia may not be suspected. For example, in one of Lohr's[6] well established and thoroughly studied cases with true polycythemia the counts and hemaglobins were normal on isolated occasions. Again (as was the case during our patient's first admission), the elevated erythrocyte count and hemaglobin percentage may not be supported by other findings confirmatory of the diagnosis of true polycythemia and one may not feel justified in making such a diagnosis. However, it is coming to be more and more recognized that a latent or unrecognized polycythemia vera may be present in obscure cases of peripheral vascular disease—chronic leg ulcers, acrocyanosis, *etc.,* as pointed out by Dameshek and Henstell[11] in an excellent article on the diagnosis of polycythemia. So, until the condition of "effort thrombosis" becomes more clearly understood we believe a thorough search should be made in every case for polycythemia.

Pathology.—With the overabundance of erythrocytes and with hematocrit values well above normal, the viscosity of the blood in polycythemia vera is increased and the rate of flow probably reduced. Add to these predisposing conditions some precipitating factor and thrombosis results. (This may well be some slight trauma or, again, as here, no exciting cause may be traceable.) Just where the thrombosis occurs is not always detectable, and our rather general title "Thrombosis and Gangrene of the Right Arm, *etc.*" is used purposely. In fact, we feel that specific designations of axillary or subclavian thrombosis should be reserved solely for those cases where a demonstrable thrombosis can be found in the vein named and should not be used for general swelling of the arm following a supposed thrombosis which is assumed to be high because the swelling extends to the shoulder or trunk. When thrombosis does set in there follows a marked circulatory disturbance in and about the area drained by the affected segment. A generalized swelling comes on and lymphatic blockage is evident. This may rest upon the Leriche[12] principle of arteriolar spasm, as suggested specifically in thrombosis of the arm by Cattalorda,[13] Roelsen,[14] and by Ochsner[15] in the case of acute phlebitis of the leg, and may lead to secondary edema, lymphedema and stasis. In fact, it is suggested by Cattalorda that the whole process may be one of vessel spasm and that no thrombus is necessary to initiate the lesion. Such feeling has arisen from the fact that in some instances axillary and subclavian veins have been exposed with the

intent of excising the affected segment and in some of these there was found no thrombosis at all. Cattalorda had one such experience. Moure and Martin[16] report the same thing, and Roelsen tells of seven cases, three of which were operated upon, but in none of these was anything abnormal found. He feels the whole process may be explained as the result of spasm alone, and regards thrombus formation as a complication. The reasoning which localizes the pathology in the subclavian or axillary veins may grow from a fallacious underlying premise of "high swelling-high thrombosis." Is it not possible that in those cases where the explored axillary or subclavian veins were found to be normal that the process did not rest on spasm alone but followed an undiscoverable thrombosis of a vein in the lower arm, forearm or wrist? In our case, when all the secondary manifestations had cleared away, the blockage was clinically demonstrated to be from the midforearm down, yet the swelling in the early stages had extended even over the chest wall. Had our case not gone on to the stage of tissue destruction but had subsided like many others, we, too, would probably be reporting a case of "subclavian vein thrombosis." Venography may prove the means of solving some of these questions.

Many careful studies have been made to explain the true thromboses which occur in the subclavian and axillary veins on an anatomic basis (Willan,[17] Gould and Patey,[18] Lohr,[6] Lowenstein,[19] and Kaplan.[20] Most of these studies lead to a theory that overstretching with compression and contusion of the vein between clavicle and first rib explains the situation (the subclavius muscle or costocoracoid ligaments exerting the pressure). Some feel the subclavio-axillary valve is damaged. Writers do not stress the rôle of the lymphatics. None points out that there may be a relationship between the fact that this lesion most often affects the upper right extremity, and that the lymphatic system draining the upper right-quarter of the body is constituted differently than the other lymphatics. One thinks of the "right lymphatic duct" receiving the tributaries of the region and emptying into the subclavian vein. Yet, in a definite percentage of cases the three main tributaries of this duct—jugular, mediastinobronchial and subclavian—do not unite to form it but empty separately into the venous system. In such cases blockage of one such main tributary would be less likely to extend to the others and would show itself by the clinical phenomenon of swelling limited to the part drained by the affected trunk. This we think may have occurred in our patient, for the whole area involved was drained by the subclavian lymphatics—the arm, obviously, the chest wall by the channels accompanying the superior thoracic, long thoracic, *etc.* Even the lymphatics accompanying the external jugular vein, so prominently involved here, follow this vessel to the subclavian lymphatics and are not a part of the internal jugular system of lymphatics. This would account for the pronounced cord-like swelling along the external jugular without swellings of the other parts of the neck, or face as seen in our patient. What the mechanism of this lymphatic

blockage is we cannot say absolutely, but it does seem to be the major cause of the alarming swelling. Microscopic sections made of the radial artery and veins in our case (Fig. 9) show definite subacute and chronic inflammatory changes in the periarterial and perivenous tissues.

As to the gangrene formation, it was due, no doubt, to the backing up of resistance to the flow of arterial blood until tissues no longer received adequate nourishment. The gangrenous process then extended up the arterial tree a certain distance, aided, we feel sure, by the arteriosclerosis present. That the original thrombosis was, in itself, massive enough to accomplish all this is unlikely but that a pernicious progressive occlusive process was initiated is more plausible. Collateral vessels were probably narrowed by spasm, their own blood supply impeded through the pressure of the lymph-edema on their vasa vasorum and, finally, their sluggish stream came to the clotting point. The mechanics of "wet gangrene," a subject not greatly discussed in recent years, is adequately dealt with by Audier and Hamorici.[21]

Symptoms and Signs.—The symptoms in our case were very similar to those in the Mayo case—painless swelling, slight tenderness, deep red cyanosis, with gradual extension of the process to the shoulder and chest wall were common to both. Cord-like prominence of the veins mentioned by Stover and Herrel[1] was noted only in the external jugular by us. Pulses at the wrist remained normal until the gangrenous process shut them off. Writing of axillary and subclavian vein thrombosis, Veal[9] gives as typical the following signs and symptoms no matter what the underlying cause: Pain in the arm and shoulder (not definite in our case); massive pitting edema of the entire extremity; weakness and partial loss of function of the arm; preservation of the radial pulse; elevation of the systolic pressure on the affected side (in ours pressures were equal); palpable, tender cord-like swelling along the course of the brachial, basillic and axillary veins (external jugular here); and marked elevation of the venous pressure and a decrease in the oxygen content of the venous blood of the affected arm (these latter two points not checked in our case).

We do not presume to discuss the diagnosis of the general disease—polycythemia vera—other than to stress the fact that not all signs are present in every case, and if one finds a persistently high erythrocyte count and hemoglobin value it may be practical to assume that the disease is present. Splenic enlargement, for example, was not demonstrable in our patient while he suffered his thrombosis in the right arm but was present a few months later. Increase in platelets was not found at the height of his red cell increase. The fragility of the cell was within normal limits—the described increased resistance to hemolysis not being shown. Yet the autopsy findings of splenic enlargement, terminal coronary thrombosis and red bone marrow hyperplasia firmly establish this as a true case of Vaquez's disease. One of the more recently discussed[22] causes of pseudopolycythemia vera—sclerosis of pulmonary vessels—was eliminated here by a chest roentgenogram which showed none of the characteristic sclerosis.

Treatment.—What has been said about the diagnosis of the general condition has a direct bearing on treatment. Although injection of the sympathetic ganglia, excision of affected vein segments, paravenous and periarterial sympathectomies have been tried, it is rather generally agreed (Veal, Matas, Roelsen) that the treatment of thrombosis is supportive—elevation, rest, moist warm applications, *etc.* In other words, one does little but promote circulation and await the reestablishment of ample venous return through collateral channels. But in our case this was not enough, probably because of the markedly increased viscosity and slowing of flow which accompanies polycythemia vera, and which tends to promote an extension of the thrombus formation into those channels which ordinarily might serve as detours (Fig. 8). In the Mayo case, prompt recognition of the general disease and ample blood-letting (5,250 cc. over an eight-day period) contributed to the saving of the limb. Perhaps (although we cannot be sure) this procedure would have resulted in a better outcome in our patient. However, in our own defense, we must point out that Lohr[6] had two cases of polycythemia vera with the thrombosis syndrome in the arms, and in neither case did gangrene result even though the treatment was entirely "supportive." The point of difference was that these two patients were young and otherwise healthy, while our patient was old and markedly sclerotic.

If gangrene is inevitable, it is wise, we believe, to await the line of demarcation and then amputate. The way our flaps healed, despite an already present infection, showed that circulation to the line of tissue change was quite competent.

Liability.—Of practical importance from another standpoint is the interesting fact that "effort thrombosis" coming on after trauma—even the slightest—suffered at work has been adjudged a compensable accident. Louisiana established this precedent, the courts submitting the question for decision to Dr. Rudolph Matas, who came to this conclusion after a thorough study of the subject. If there was no preceding trauma, however, Matas felt no liability existed. Since the question of the presence of a predisposing factor would be of great importance in settling such a question, we feel that here is one other reason for making a thorough search for polycythemia vera in all cases of so-called "effort thrombosis."

SUMMARY AND CONCLUSIONS

1. A case of thrombosis in the vessels of the arm accompanied by massive swelling extending to the chest wall and neck in a patient with polycythemia vera is reported.

2. Seven other such cases have been traced in the literature. Our case was the only one in which the process resulted in gangrene.

3. We believe, as did Lohr,[6] that an unrecognized polycythemia vera may underlie a certain number of cases of so-called "effort thrombosis." It should be carefully sought for in each future case to prove or disprove this contention.

4. Thrombosis of vessels in the lower arm, forearm or wrist, plus the extensive lymphatic stasis which follows, may explain many cases incorrectly designated as examples of subclavian or axillary thrombosis.

5. In a patient suffering from a thrombosis, increased red cell and hemaglobin values call for therapeutic blood-letting, regardless of whether the condition can be accurately diagnosed as a true polycythemia or not.

6. Since the question of compensation may arise in cases of "effort thrombosis" it is well to establish the presence or absence of polycythemia vera in such cases.

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IMMEDIATE AND LATE RESULTS OF PERFORATION OF PEPTIC ULCER

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IN THE 15-YEAR PERIOD, from 1925 to 1940, there have been 57 cases of acute perforation of peptic ulcer admitted to Vanderbilt University Hospital, of which 51 were in the duodenum and six in the stomach.

This study was primarily for the purpose of testing the validity of the widespread opinion that if a peptic ulcer perforates, and if the patient recovers after closure of the perforation, subsequent permanent healing of the ulcer may be expected to occur. This opinion constituted the basis for a method of treatment of peptic ulcer described by Balfour in 1918. Balfour states: "Complete perforation by the cautery point through the center of the crater of the ulcer has been made an essential in the technic because of the clinical fact (drawn attention to early by Mayo, Clairmont and others and now generally recognized) that spontaneous and complete perforation of a gastric ulcer is, presupposing recovery from this accident, quite likely to be followed not only by the cure of the ulcer but by the cure of the patient." In recent textbooks on surgery one finds some difference of opinion as to late results obtained in the patients in question. Christopher has said: "Healing of the ulcer occurs after simple purse-string suture in about three-fourths of the cases." More recently, Cutler has written: "After three to four days the patient is started on strict medical regimen, which may be continued for an indefinite period, because simple closure of the perforation has not cured the patient of his ulcer or of his tendency to form another." And, again, Horsley has written: "Frequently after closure of the perforation the lesion appears to heal and the patient remains well, but if symptoms continue, as they do in many cases, the patient can be treated anew as a peptic ulcer case that demands surgery." Grey-Turner estimated that only 50 per cent of their patients with perforations were cured after recovery from the perforation. Lewisohn found that 39 per cent of his patients had persistence of ulcer symptoms. Sullich reported that 70 per cent of his patients were not cured by perforation.

SUMMARY OF CASES

The operative procedure employed in the present series of 57 cases consisted, essentially, in removal of all available foreign material in the peritoneal cavity followed by simple closure of the perforation either by purse-string or Lembert sutures.

In three cases the perforation of the ulcer occurred as the first indication to the patients of the presence of gastro-intestinal disorder. Sixteen patients had had symptoms of peptic ulcer for ten years or more prior to the abdominal

No. of Cases	Av. Age	Av. Duration of Ulcer Symptoms	Amount of Fluid in Peritoneal Cavity	Wound Infection and Disruption	X-ray Evidence of Air	Size of Perforation	Deaths
			37 large*	12 infections	38+	10 large†	
57	41 yrs. (14–70)	6.3 yrs.	18 small 2 not indicated	4 disruptions	11− 8—no x-ray	47 small	16 (28.07%)

* Large—greater than 1,000 cc.
† Large—greater than 1 cm.

catastrophe for which they sought admission to the hospital. None of the patients had adhered to a strict medical regimen of therapy for the ulcer, only eight having had even a short course of formal therapy. The remainder had taken alkali or a bite of food irregularly for relief of abdominal distress.

All the patients gave a history of sudden onset of severe abdominal pain of the nature usually found in this condition. Pain was usually soon followed by vomiting; only eight patients noticed the presence of blood in the vomitus or gave any previous history of either hematemesis or tarry stools. Abdominal rigidity was the rule on examination, but liver dulness was obliterated or impaired in less than one-half of the cases.

No correlation was found to exist between the size of the perforation and the volume of fluid or the presence of air in the peritoneal cavity. Again, no correlation was found between the lapse of time from the time of perforation to the time of operation and the amount of fluid in the peritoneal cavity. The incidence of wound infection increased with the lapse of time between perforation and operative intervention. There were, also, four wound disruptions which showed no relation to lapse of time between perforation and operation or to the occurrence of infection.

The cases have been arbitrarily divided into three groups, according to the number of hours which elapsed from the time of onset of symptoms of perforation to the time of operation.

Group	No. of Cases	Av. Age	Av. Duration of Ulcer Symptoms	Amount of Fluid in Peritoneal Cavity	Wound Infection and Disruption	X-ray Evidence of Air	Size of Perforation	Deaths
				18 large*	5	20+	25 small	
A						7−		4
0–10 hrs.	30	40 yrs.	5.8 yrs.	10 small 2 not indicated	2 disruptions	3—no x-ray	5 large†	(13.3%)
B				8 large	3	9+	9 small	
10–20 hrs.	12	44.7 yrs.	6.5 yrs.		2 disruptions	2−		5
				4 small		1—no x-ray	3 large	(41.6%)
C				11 large		9+	13 small	
20+ hrs.	15	40 yrs.	6.9 yrs.		4	2−		7
				4 small		4—no x-ray	2 large	(46.6%)

* Large—greater than 1,000 cc.
† Large—greater than 1 cm.

On comparing these groups, one immediately sees that the mortality rate jumps from 13.3 per cent in cases operated upon before ten hours had elapsed

to 45.5 per cent in cases operated upon later than ten hours. No wound infections or disruptions occurred in patients operated upon under three hours.

GASTRIC ULCER

Case No.	Age	Duration of Symptoms of Ulcer	Duration of Perforation	Signs of Air	X-ray Evidence of Air	Size of Perforation	Wound Infection of Disruption	Duration of Hospitalization	Symptoms Since Operation
72810	23 yrs.	1 mo.	24 hrs.	+	+	Small	0	12 days	+
79346	51 yrs.	16 yrs.	14 hrs.	+	+	Large	0		Expired day of operation
88151	45 yrs.	4 yrs.	21 hrs.	+		6 mm.	0	11 days	+
93969	53 yrs.	6 yrs.	12 hrs.	+	+	1.5 cm.	Infection	18 days	Died
356	36 yrs.	4 yrs.	10 hrs.		−	2 mm.	Disrupted, 7th Day	30 days	Asymptomatic 3 yrs. Symptoms 12 yrs.
95333	20 yrs.	1 yr.	2½ hrs.			2 mm.	0	14 days	+

There was no history of hematemesis in any member of this group as contrasted with the cases of duodenal ulcer, in which there were eight patients who vomited blood prior to operation. The patients were all males, averaging age 38.

Since Vanderbilt University Hospital draws its clientele from a large rural area, considerable time elapsed in many cases before the patients presented themselves. Nine of the patients who died postoperatively were seen by their local physicians within a few hours of the time of perforation; were given hypodermic injections; and were not advised to go to the hospital until the following day or later. Of the 57 patients operated upon, 41 left the hospital, and 16 died, a mortality rate of 28.6 per cent as compared to the operative mortality of a collected series of 15,340 cases reported during the last decade of 23.4 per cent (DeBakey). Bager, in reviewing 1,495 cases, found the mortality to be 14.7 per cent for the first six-hour period; 26.5 per cent for the second six-hour period; 47.2 per cent for those operated upon between 12 and 24 hours after perforation; and 65.6 per cent for those operated upon after 24 hours.

The average age of the 16 patients dying was 43.3 years, or approximately the same as that of the group as a whole. In all 16 of these patients a large amount of fluid and gastric content was found in the peritoneal cavity at the time of operation. The amount and time of ingestion of food prior to operation was the most important factor contributing to the amount of fluid in the peritoneal cavity at the time of operation, pyloric obstruction apparently playing no important part in its causation.

In ten patients operated upon within three hours or less after perforation, three patients expired; all three of these perforations occurred while the patients were in the hospital. Also, in all three of these patients there occurred hemorrhage from the ulcer shortly before or at the time of perforation. Two of these cases had been admitted to the Medical Service, one because of

bleeding and one because of an increase in the epigastric pain of one week's duration. The third was admitted for a dilatation of a stricture of the anus, which was incident to a hemorrhoidectomy, performed elsewhere, one week previously. In Group A there was only one other patient with history of either hematemesis or tarry stools. This patient was operated upon five hours after perforation, and he too was found to have a large amount of bloody fluid in the peritoneal cavity; likewise the outcome of his case was fatal. These four cases comprised the total deaths occurring in the patients operated upon before the expiration of ten hours after perforation. As shown, there was concomitant hemorrhage in each of these cases. Regarding these four cases, it should also be noted that the perforations varied in size from 1 to 3 cm. in diameter.

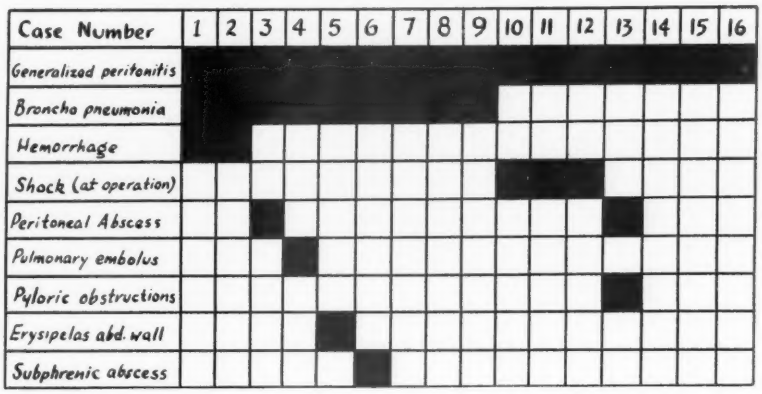

CHART I

Aside from the three patients mentioned above, there have been two others who perforated while in the hospital, both during gastro-intestinal roentgenologic examinations. Both of these had neither hemorrhage nor obstruction, and both made an uneventful recovery. One of these is a recent case who is not included elsewhere in this study.

In the entire series of 57 patients, there were only eight who gave history of either hematemesis or of tarry stools prior to operation. Three of these have just been discussed; a fourth case was the patient who was mentioned elsewhere herein, who suddenly perforated a second time, seven years after the first operation. Two more of these patients recovered satisfactorily from the operation but have continued to have ulcer symptoms, without, however, additional bleeding. A seventh member of this group who had recognizable bleeding prior to operation expired, but he was one of those who came to the hospital on the second day following perforation. The eighth member of this group recently returned to the hospital, one year after operation, with a massive hemorrhage as the first indication of persistence of his ulcer. From

these facts it seems clearly evident that the prognosis in peptic ulcer is gravely affected by hemorrhage as concerns both mortality and morbidity.

DEATHS

Of the 16 patients who died, postmortem examination was performed in nine. Dual ulcers were found in the duodenum in three of these nine patients. These ulcers were all of the so-called "kissing" variety, in each instance one occurring in the anterior and one in the posterior wall of the duodenum; the perforation was in the ulcer on the anterior wall of the duodenum in each case; in none of these cases had recognizable hemorrhage occurred, either prior to operation, at operation or at autopsy. In another of the autopsied cases, an open artery was found in the margin of the ulcer, which was located on the anterosuperior wall of the first portion of the duodenum.

Course Subsequent to Hospitalization.—All of the 41 patients who recovered following operation were traced. Two of these had expired, one of them two and one-half months postoperatively, the cause of death apparently being residual peritoneal infection complicated by empyema; the other patient lived two years, continued to have symptoms, and died of intercurrent infection (pneumonia). Of the remaining 39, seven patients (17.5 per cent) report themselves to be completely free of gastro-intestinal symptoms, while 32 of them (82.5 per cent), including all four of the cases of gastric ulcer who survived, continued to have varying degrees of symptoms of peptic ulcer. These symptoms consisted, essentially, in epigastric pain relieved to some extent by food or alkali, occasional nausea and infrequent vomiting. Very few of these patients have followed strictly any course of medical treatment since operation. Seven of these 32 patients had symptoms ever since operation, while 25 of them have had an asymptomatic period immediately following operation varying from three weeks to six years, averaging 1.8 years. It is interesting, and perhaps informative, to note that the patient reporting the longest asymptomatic period had this period abruptly terminated, without warning, by second perforation of his ulcer.

The seven patients recorded above as being asymptomatic have been so for the following number of years, respectively: seven, seven, four, one, eight, twelve and eight years.

Subsequent Roentgenologic Studies.—Sixteen patients out of the 41 who survived have had roentgenologic examinations subsequent to operation, varying in time from 15 days to seven years postoperatively. In every case except one there was found a deformity of the duodenal cap. No distinction can always be made between simple postoperative deformity and that due to a persistent ulcer. The single patient whose gastro-intestinal series was normal had been on a strict ulcer regimen for the eight months preceding the examination, and subsequently he has had a recurrence of his old ulcer symptoms. In three of this group there was some roentgenographic evidence of duodenal

obstruction, which was in agreement with the clinical picture of the cases in question.

SUMMARY AND CONCLUSIONS

(1) Fifty-seven cases of acute perforated peptic ulcer are presented.

(2) The relation of the duration of perforation prior to operation and the hospital course is discussed.

(3) The cause of death and the findings at autopsy are outlined.

(4) The concomitant occurrence of the complication of perforation and bleeding was found to alter greatly the mortality and morbidity.

(5) None of the patients had received adequate medical treatment for ulcer.

(6) The incidence of recurrence of symptoms of ulcer postoperatively, after simple closure of the perforation, was found to be 82.5 per cent.

(7) Following recovery from the operation for closure of a perforated peptic ulcer, the patient should be evaluated anew and treated just as any other case of peptic ulcer.

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THE LYMPHATIC AND VENOUS SPREAD OF CARCINOMA OF THE RECTUM*

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AN UNDERSTANDING of the spread of carcinoma of the rectum is essential for the treatment of the disease. Four main routes are possible: (1) By direct extension; (2) by the lymphatics; (3) by the blood stream; and (4) by transplantation through the peritoneal cavity. The second and third routes of spread are the subjects of this report.

LYMPHATIC SPREAD

The clinical observations of Miles,[1, 2] in 1925, on the lymphatic spread of carcinoma of the rectum and injection studies by French and German anatomists have formed the basis for its present surgical treatment. Recent work by Gilchrist and David,[3] and Coller, Kay, and MacIntyre[4] has thrown further light on the subject. The present study makes use of their methods, and is based on the examination of 75 specimens of the rectum and rectosigmoid removed at operation during the past three years. Sixty-two were removed by abdominoperineal resection, ten by perineal excision, and three by anterior abdominal resection of the rectosigmoid, with either colostomy and inversion of the distal stump or end-to-end anastomosis. In these three cases the superior hemorrhoidal vessels were divided and a wide area of the mesentery was removed. All specimens were cleared by the Spalteholtz method, as used by Gilchrist and David, with some slight modifications. All lymph nodes were dissected out, sectioned, and charted on drawings to show their relationship to the tumor and the main blood vessels. By this method, many more lymph nodes were found than would have been possible by less accurate methods. As a result the number of node metastases found was also greatly increased. Metastases were frequently seen in the smallest nodes, with a diameter of only 1–2 mm. Mere size proved to be a completely unreliable guide to the presence or absence of a metastasis in a node.

In this series of 75 cleared specimens, the average number of nodes per specimen was 52. The average number found in specimens removed by abdominoperineal resection was 54, by perineal proctectomy, 31, and by anterior resection, 67. The greatest number found in any one specimen was 124. Metastases were found in 55 per cent of the cases. This incidence is somewhat less than that of 68 per cent reported by Gilchrist and David[3] in 25 cleared cases, and that of 64 per cent found by Coller[4] in 53 cases. It stands out in contrast, however, to that of 36 per cent which was found in a similar series[15] of cases at this hospital, from 1916–1932, in which the clearing tech-

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nic was not used. The incidence in the present series is 19 per cent higher, and demonstrates the value of this method. This increase occurred in spite of the fact that more cases in this series were operated upon at an earlier stage, when fewer metastases might be expected. Thus, in the 1916–1932 series, 34 per cent of the specimens examined grossly were completely annular, whereas in the present series only 22 per cent were annular. That a relationship exists between the extent of bowel circumference involved and the frequency of metastasis will be shown later.

TABLE I

INCIDENCE OF NODE METASTASES REPORTED BY PREVIOUS AUTHORS

	No. of Specimens	No. of Nodes per Specimen	Per Cent with Metast.
McVay	100	6	47%
Wood and Wilkie	100	11	51%
Westhues	74 (cleared)	25	59%
Gabriel, Dukes, and Bussey	100	28	62%
Gilchrist and David	22	24	68%
	25 (cleared)	52	
Coller, Kay, and MacIntyre	53 (cleared)	67	64%
Grinnell (1916–1932)	107	—	36%
Present series (1938–1941)	75 (cleared)	52	55%

THE LYMPHATICS OF THE RECTUM.—Our knowledge of the lymphatics of the rectum is based chiefly on the injection studies of Delamere, Poirier, and Cuneo,[5] Villemin,[6] and Rouviére.[7] The intramural lymphatics which lie in the mucosa, submucosa, and muscle layers, are continuous with those in the rectosigmoid above and with the dermal and subcutaneous lymphatics of the anus below, and drain into the extramural lymphatic system. This system consists of three main lymphatic trunks, the inferior, middle and superior, which correspond, in general, with the inferior, middle, and superior hemorrhoidal vessels, and constitute the three zones of spread—upward, lateral, and downward, described by Miles.[1, 2]

The inferior trunks arise from the anal region in the lowest portion of the rectum, and drain, chiefly, to the inguinal nodes by way of the perineum and the inner side of the thigh, and probably, occasionally, directly to the iliac and sacral nodes in the pelvis. The middle trunks arise in the rectum near the level of, and just above, the insertion of the levator ani muscles and pass laterally between the peritoneum and the levator muscles to the hypogastric and sacral nodes along the middle hemorrhoidal and sacral vessels. The superior trunks arise from the entire length of the rectum from as low as the anal canal and drain into perirectal and mesocolic nodes along the superior hemorrhoidal and inferior mesenteric vessels. As these three lymphatic trunks anastomose with each other, it is possible for a cancer of the rectum to have metastases along two or even three routes of spread.

In none of our cases was extensive intramural lymphatic spread in the submucosa or muscularis found. No attempt was made, however, to study this point by serial sections. We are inclined to agree with Miles,[1] Wood and Wilkie,[8] Westhues,[9] Cole,[10] and Monsarrat[11] that this type of spread is

rare. As far as intramural extension is concerned it is probably safe to divide the bowel within a few centimeters of the tumor.

Upward Spread.—The extramural lymphatic spread in the 41 cases with lymph node metastases in our series of 75 rectal and rectosigmoid carcinomata, was studied carefully. The main lymphatic path is upward along the superior hemorrhoidal vessels. In over one-half of the cases with node metastases only three nodes or less were involved. The largest number of

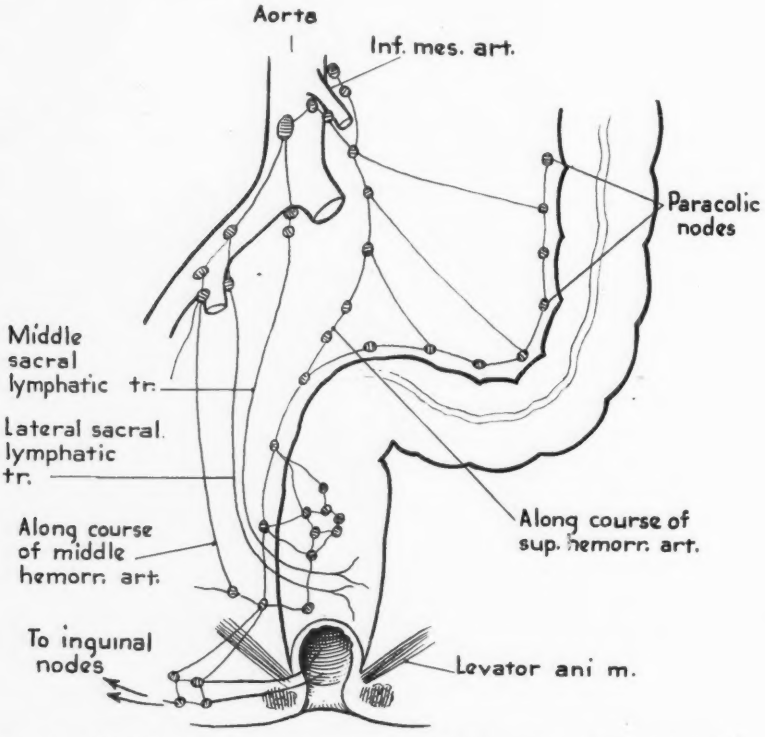

Fig. 1.—Schematic drawing of the extramural lymphatic system of the rectum and pelvic colon (after Miles and Villemin).

metastases in any one specimen was 13. In over one-half of these cases all the involved nodes lay within 3 cm. of the tumor. If cases without metastases are included, 73 per cent of all cases in this series were found to have either no metastases or very localized ones. In the remaining 20 cases, or in nearly one-half of those with metastases, some of the involved nodes were at a considerable distance from the tumor. One can conclude that the disease tends to remain localized and usually spreads slowly through the lymphatics but that exceptions to this rule are frequent.

In nine, or 22 per cent, of the 41 cases with metastases the most proximal nodes near the point of division of the superior hemorrhoidal vessels showed metastases. This group would be expected to have a particularly unfavorable prognosis. Gabriel, Dukes, and Bussey[12] found that 30 per cent of their

cases with metastases were in this group. Five of our nine cases with the most proximal node involved, had had an abdominoperineal resection, and

TABLE II

NODE INVOLVEMENT IN 41 CASES WITH METASTASES

Cases with 1 node involved	11
Cases with 2 nodes involved	2
Cases with 3 nodes involved	10
	23
Cases with 4 or more nodes involved	18
	41

four had had a perineal excision. Thus, only 8 per cent of 62 cases having an abdominoperineal resection fell into this unfavorable group, whereas 40 per cent of our ten cases having a perineal excision were so classified. This emphasizes, strikingly, the inadequate removal of lymph node-bearing tissue obtained by the perineal operation.

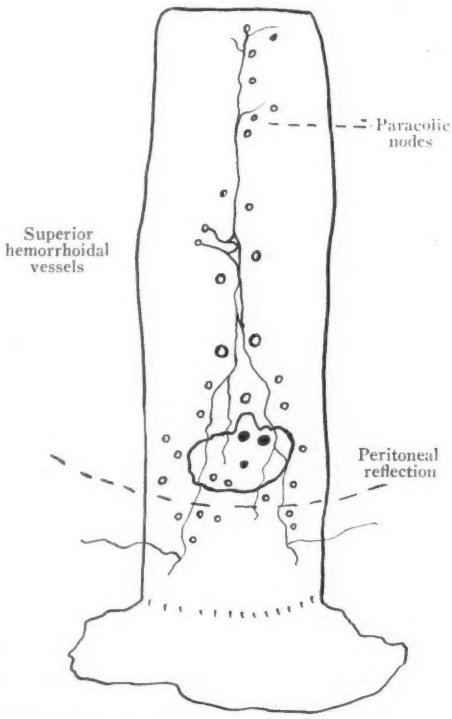

FIG. 5.—A carcinoma with three metastatic nodes within three cm. of the tumor. For the sake of clarity not all uninvolved nodes are charted.
O = nodes without metasases.
● = nodes with metastases.

In most of our cases the lymphatic spread of the disease progressed upward along the superior hemorrhoidal vessels in a fairly orderly manner. In seven of our 41 cases with metastases, however, the spread was definitely discontinuous, with a considerable gap of uninvolved nodes between the tumor and the more proximal involved ones. Figure 5 shows such a specimen. Gabriel, Dukes, and Bussey[12] had only one such case in 62 cases with metastases, whereas Wood and Wilkie[8] reported six in 51 cases. This discontinuity of spread is undoubtedly explained by the fact that there are a variety of upward lymphatic pathways. In injection studies, Villemin, Montagné, and Huard[6] describe three lymphatic routes passing upward with the superior hemorrhoidal vessels. The short paths are the most numerous and drain into a group of nodes near the bifurcation of these vessels. The middle paths pass upward without stopping at intermediate nodes to enter nodes

near the junction of the superior hemorrhoidal and lowest sigmoid vessels. The long paths pass upward without any intermediate stops to enter nodes near the junction of the left colic and the inferior mesenteric vessels. In six of our cases one or more nodes at the bifurcation of the superior hemorrhoidal vessels were involved, and in four cases nodes at the junction of the superior hemorrhoidal and lowest sigmoid arteries showed metastases. Two of these

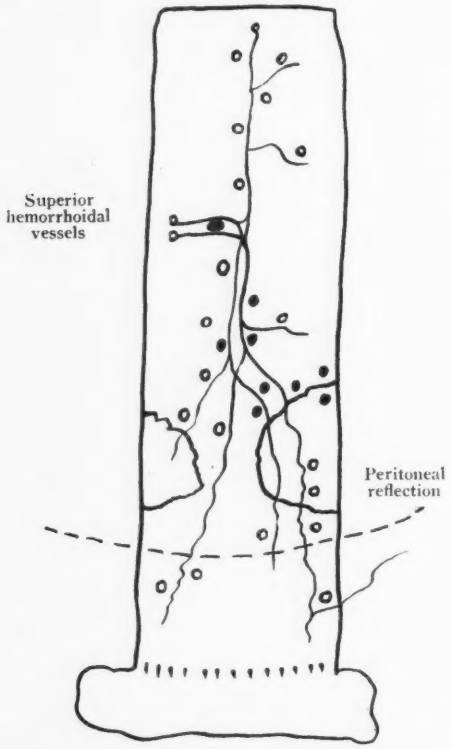

FIG. 3.—A carcinoma removed by abdominoperineal resection with a metastasis in one of the most proximal nodes near the point of ligation of the superior hemorrhoidal vessels.

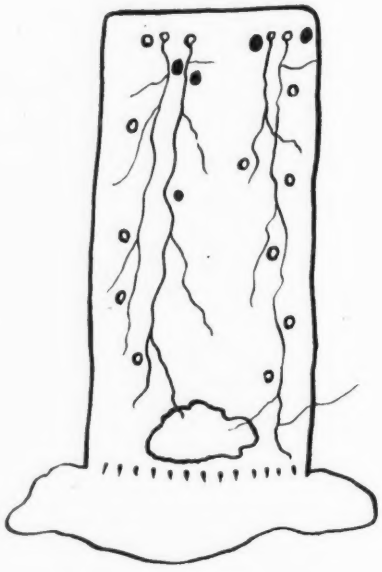

FIG. 4.—A low rectal tumor removed by perineal excision with metastases in the most proximal nodes near the divided branches of the superior hemorrhoidal vessels.

four cases, apparently, represent examples of direct spread along the middle route. The other two showed metastases at both these sites, and may either represent spread by both the short and middle routes or a secondary relay from the short route which ends at the bifurcation. We had no cases illustrating metastasis by the long route, as the usual abdominoperineal resection does not extend as high as the origin of the left colic vessels.

The cases with lymph node metastases were also studied to compare the adequacy of simple perineal excision with that of abdominoperineal resection. The average limit of adequate removal of mesentery in the perineal operation was taken to be 3 to 4 cm. above the peritoneal reflection. Only cases with tumors lying completely beneath the peritoneal reflection were taken. Some

cases with tumors partly above and partly below the peritoneum could probably have been included, but it was decided to limit the group only to cases best suited to the perineal operation. There were 17 cases with node metastases in this group. Only four of the 17 cases, or 24 per cent, appeared to be curable by perineal excision. In the remaining 13 cases, the involved nodes could not have been reached by such a limited operation. The inadequacy of this operation is obvious.

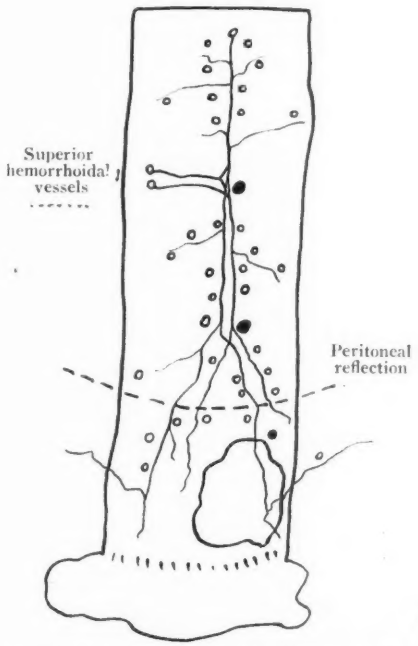

FIG. 5.—A carcinoma with discontinuous lymphatic spread. One involved node lies at the bifurcation of the superior hemorrhoidal vessels and another at their junction with the last sigmoid vessels.

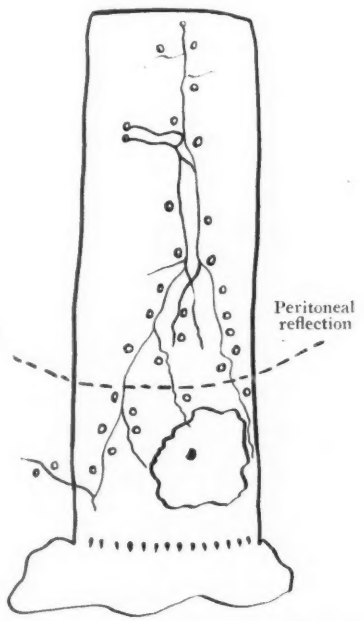

FIG. 6.—A low rectal carcinoma lying below the pelvic peritoneum. It has only one nearby metastasis and is suitable for the perineal excision operation.

LATERAL SPREAD.—The lateral spread of the disease along the lymphatics accompanying the middle hemorrhoidal vessels has been an alternate route, emphasized especially by Miles[1, 2] for carcinomata arising in the lower rectum near the level of the levator ani muscle insertion. Gilchrist and David had four cases, and Coller six, in their series. Wood and Wilkie had no instance of it. Our study showed only one case (Fig. 8), which demonstrates upward, downward, and lateral spread from a tumor situated 6 cm. above the pectinate line. It seems probable that lateral spread is most apt to occur in a low-lying tumor, where the main upward channels have been blocked by extensive metastases in the nodes. This was true in our case, and in Coller's six cases. As the middle hemorrhoidal vessels are sometimes hard to identify in cleared specimens it is possible that in one or two instances lymph nodes along this route may have been incorrectly charted along superior hemorrhoidal branches.

We agree with Gilchrist that section of the levator ani muscles should be made as widely as possible in order to reach nodes along this pathway. It seems probable, however, that this route of spread is of secondary importance, and occurs chiefly in advanced cases when the main upward path has been blocked.

Downward Spread.—Downward lymphatic spread has also been emphasized, chiefly by Miles.[1, 2] All writers on the subject agree that it occurs only when extensive metastases have occurred, blocking the other routes and causing retrograde lymph flow downward. Miles' observations were based mainly on cases with recurrent tumor nodules following inadequate perineal operations. We had only one case (Fig. 8) which has already been cited as illustrating upward, lateral, and downward spread. In this case the tumor lay

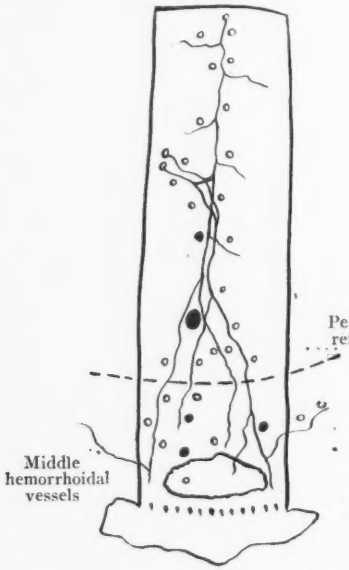

Fig. 7.—A low tumor with high node metastases unsuitable for the perineal excision operation.

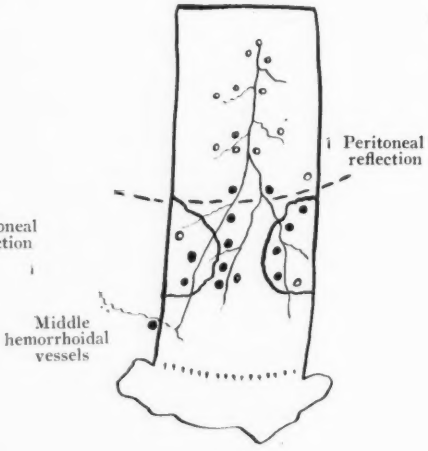

Fig. 8.—A low rectal carcinoma illustrating upward, lateral, and downward lymphatic spread. One involved node lies along the middle hemorrhoidal vessels two centimeters distal to the tumor. Thirteen of 46 nodes showed metastases.

6 cm. above the pectinate line and had metastasized to 13 nodes, 12 involved nodes blocking the upward spread and one lying along the middle hemorrhoidal vessels, about 2 cm. distal to the tumor. Westhues[8] had only one case in 74 operative resections which lay 1 cm. below the tumor and was associated with other metastases in the nodes above. Gilchrist reported one case with a metastasis 4 cm. below the tumor, and Coller another, with the involved node lying 1 cm. distal to the growth. It seems evident that downward lymphatic spread is of little importance except in advanced cases, which are probably already inoperable because of extension along the main lymphatic route upward. The very rare occurrence of downward extension in our

cases, and in those of others, suggests that the operation of anterior resection with ligation of the superior hemorrhoidal vessels may have a greater usefulness for tumors just above the rectosigmoid junction. There were three of these cases in our series with metastases, and none showed evidence of retrograde extension.

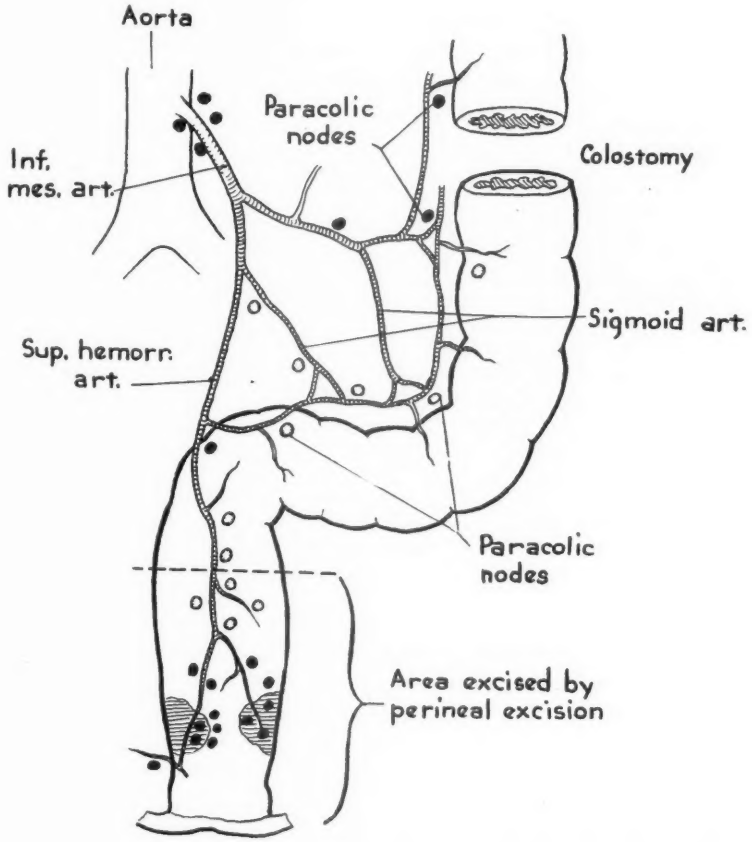

FIG. 9.—A drawing of a tumor with late retrograde extension to the paracolic nodes following perineal excision and the probable node metastases present. This is the same case as that shown in Figure 8.

Metastases to the paracolic nodes are rarely found in operative specimens. There is, apparently, little tendency for retrograde flow to take place at this stage from the inferior mesenteric-superior hemorrhoidal chain laterally to the paracolic nodes along the pelvic colon. We found no instance of it in any of our 62 specimens removed by radical abdominoperineal resection. Miles[1, 2] has stated that the paracolic nodes frequently show metastases by retrograde involvement. Wood and Wilkie[8] had no cases of it. Gabriel, Dukes, and Bussey[12] had only one such case, and this was an advanced growth with extensive involvement of the higher nodes. We had one clinical example of it, very similar to one reported in detail by Miles. This case has already been

described (Fig. 8). The patient, a female, age 55, with a low rectal tumor, had had a colostomy and a perineal excision performed in two stages. At the first operation no liver or intraperitoneal metastases were found. The tumor lay well below the peritoneal reflection. One month later the perineal excision was performed, with removal of the lower 27 cm. of the rectum. Metastases were found in 13 of the 46 lymph nodes. All the involved nodes were fairly close to the tumor. Five months later, two recurrent tumor nodules were removed from both the proximal and distal limbs of the colostomy, and showed a histologic picture similar to that of the original tumor. The nodules evidently represented retrograde spread from metastatic paracolic nodes. It seems probable that involvement of the paracolic nodes occurs late, when radical surgery can offer little hope of cure.

No relationship could be demonstrated between the size of the tumor and the frequency of metastases. In fact the square area of bowel involvement was very slightly greater in the tumors without metastases than in those with them. There was some correlation, however, between the amount of bowel circumference involved and the frequency of metastasis. Thus, the incidence of node metastases was 53 per cent when the tumor was not completely annular, and 71 per cent when it was.

VENOUS SPREAD

The occurrence of blood-borne metastases from carcinoma of the rectum has long been recognized, but the relative importance of this route of spread is still largely a matter of conjecture. It is apparent from the frequency of five-year postoperative survivals in early cases without lymph node metastases, that venous metastasis does not usually precede lymphatic involvement. Many surgeons, however, are familiar with the occasional small, early tumor, without node involvement, which is found to have liver metastases at operation. Brown and Warren[13] have recently thrown further light on this subject. They studied a series of 165 cases of carcinoma of the rectum in which complete postmortem findings were available. These were cases of patients dying immediately after resection or colostomy, survivals following operation, and cases dying of the disease without operation. These cases were studied particularly for local blood vessel invasion in the tumor and for visceral and lymph node metastasis. We have made a similar study in our series of 75 cases. The search for vessel invasion by tumor cells was carried out as recommended by Brown and Warren. At least three sections of the tumor were taken in each case and stained with Masson's aniline blue trichrome stain to emphasize the smooth muscle wall of the small veins. The chief difficulty encountered was in differentiating tumor cells in veins from those in lymphatics. The main point in differentiation was the presence of smooth muscle cells in the vessel wall not usually seen except in the largest lymphatics. The finding of red blood cells in the lumen, especially if in considerable numbers, was also helpful. Vessel invasion was found most frequently in the fat and connective tissue outside the muscle wall of the rectum along the deep

edge of the tumor. The next most common site was in the submucosa. Nearly every case was checked by Dr. A. P. Stout. Where there was any doubt of blood vessel invasion, it was not accepted.

Blood Vessel Invasion.—In the 75 specimens of carcinoma of the rectum in this series, in which blood vessel invasion was studied, definite invasion was found in 36 per cent. If 84 colon carcinomata, which were also studied, are added to this group, the incidence of vessel invasion rises to 41 per cent. Brown and Warren[13] found it in 61 per cent, but the majority of their specimens were from autopsies of advanced cases in which

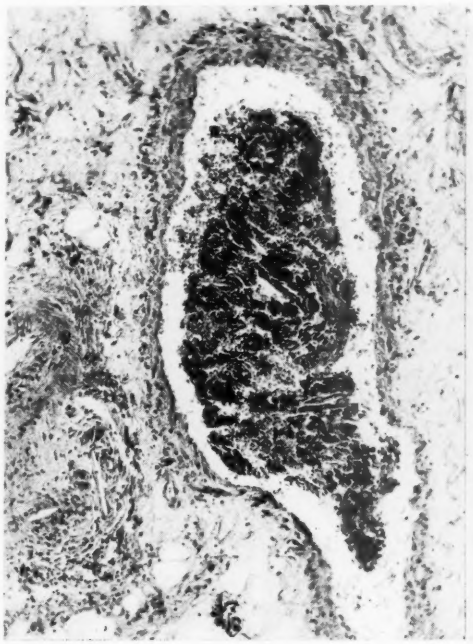

Fig. 10.—Tumor cells in a small vein. Note the accompanying artery. (x125)

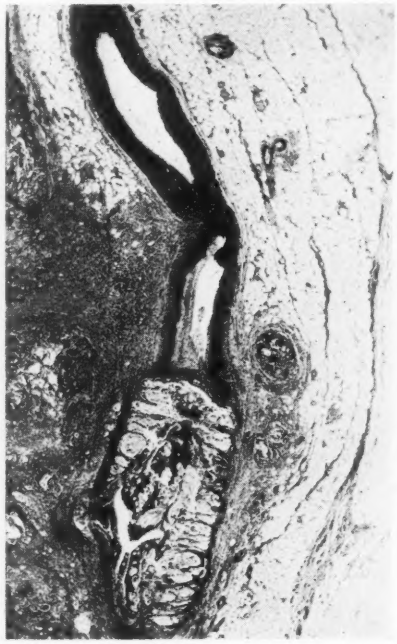

Fig. 11.—A tumor thrombus adherent to the wall of a vein. (x28)

no operation had been attempted. A higher frequency of vessel invasion might be expected in these cases. They probably represent a less differentiated and more invasive group of tumors, and one in which vessel invasion has had more opportunity to occur in the late stages of the disease.

Table III

INCIDENCE OF LOCAL BLOOD VESSEL INVASION

	Total Cases	Vessel Invasion	
		Cases	Per Cent
Present series:			
Rectum (operative specimens)	75	27	36%
Colon (operative specimens)	84	39	46%
Brown and Warren:			
Rectum (operative and nonoperative specimens)	165	100	61%

The frequency of vessel invasion was found to vary with the degree of differentiation of the tumor. The growths were classified into three grades —I, II, and III; using as criteria the degree of invasive tendency, glandular arrangement, nuclear polarity, and the frequency of mitoses. This classification has been described previously.[15] Our series of 75 cleared specimens was increased by 15 other rectal carcinomata from a 1916–1932 group, which were also searched for tumor invasion in the veins. These additional cases had little effect on the result, but contributed to a larger series of cases. Twenty-five per cent of the Grade I cases had vessel invasion, 34 per cent of the Grade II cases, and all of the Grade III cases. That such a relationship should exist is not surprising. Just as poorly differentiated tumors frequently invade nerve sheaths, so do they often penetrate into the lumen of vessels. We have already shown previously[15] the close relationship between the grade of the tumor, the frequency of node metastasis, and the five-year results. The value of grading is further demonstrated here. It influences prognosis, whether the disease spreads by the lymphatics or by the blood stream.

TABLE IV

VESSEL INVASION AND HISTOLOGIC GRADE

	Grade I	Grade II	Grade III
Vessel Invasion:			
Absent:			
Cases	12	38	0
Per cent	75%	66%	—
Present:			
Cases	4	20	16
Per cent	25%	34%	100%

It was also found that the frequency of vessel invasion increased with the depth of penetration of the bowel wall by the tumor. The cases were grouped according to Dukes'[14] classification. A-cases are those in which the growth is confined to the wall of the rectum, B-cases those which have penetrated to the extrarectal tissues but have not reached the lymph nodes,

TABLE V

VESSEL INVASION AND DUKES' CLASSIFICATION

	A	B	C
Vessel Invasion:			
Absent:			
Cases	11	20	19
Per cent	84%	69%	40%
Present:			
Cases	2	9	29
Per cent	16%	31%	60%

and C-cases those which have metastasized to the regional nodes. Vessel invasion was found to be nearly four times as frequent in C-cases as in the A-cases, of which there were two. Presumably, the tumors which have penetrated farthest are usually the least differentiated and the most invasive.

They also have a greater opportunity for vessel invasion and a larger area available for its search. In addition to the two A-cases mentioned, vessel invasion was also found in a third case, a small, early carcinoma developing on a polyp. This was a very undifferentiated Grade III tumor, which was only excised locally, and was not included in this series of cleared specimens. This patient is still well, two years after operation.

VISCERAL METASTASES.—Except in a few instances, it was not possible in this series to correlate the finding of blood vessel invasion with the later development of blood-borne metastases, because a sufficient follow-up period has not yet elapsed. Brown and Warren[13] have shown in their cases that 67 per cent of those with local intravascular invasion had visceral metastases. They emphasize the fact that many tumor cells that enter the venous circulation fail to survive and grow in distant foci. The size of the vein involved is also an important factor, as Willis[16] has pointed out. Large veins containing tumor cells are far more likely to have distant metastases than microscopic ones. In only seven of our cases had visceral metastases occurred at the time this report was made. However, 25 other cases of rectal carcinoma were found in an older series of cases,[15] from 1916–1932, which had had definite visceral metastases, and whose specimens could be sectioned and searched for vessel invasion. All but three cases had liver metastases, with occasional lung and bone involvement as well. The presence of liver metastases was determined by autopsy findings, obvious metastases seen at operation, or clinical evidence of a rapidly enlarging nodular liver.

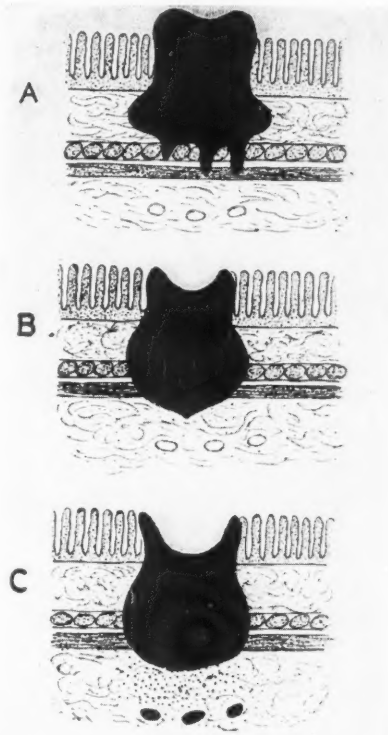

FIG. 12.—Dukes' classification of carcinoma of the rectum (after Dukes).

There were 32 cases with visceral metastases in this combined group. When the distribution of these cases into three histologic grades was studied, and compared to that of our recent cleared cases, a much larger percentage of Grade III cases was found in the former group. As we have already

shown that vessel invasion is more frequent in Grade III cases, it is not surprising to find that visceral metastases are also more frequent.

TABLE VI

DISTRIBUTION OF CASES BY HISTOLOGIC GRADE IN 32 CASES WITH VISCERAL METASTASES AND IN PRESENT SERIES

	Grade I	Grade II	Grade III
Cases with visceral metastases selected from present and 1916–1932 series:			
Cases	4	15	13
Per cent	13%	47%	40%
Present series (all cases):			
Cases	15	52	13
Per cent	19%	65%	16%

If this combined group with visceral metastases is analyzed as to bowel wall and node involvement, as measured by Dukes' classification, several striking facts appear. In the first place, no A-cases were found in this group. Evidently venous metastasis did not occur until after the tumor had completely penetrated the muscle wall of the rectum. Secondly, the percentage of B-cases, *i.e.*, cases with complete tumor penetration of the muscle wall, but without node involvement, with visceral metastases was surprisingly high and will be discussed further in the next paragraph. Finally, the proportion of C-cases, *i.e.*, cases with complete tumor penetration of muscle wall and node metastases, was found to be large, as might be expected. The comparison of this group with our recent cleared cases, and also with the 1916–1932 series, emphasizes these points. The farther the tumor penetrates the bowel wall the more frequent is vessel invasion and the chance of blood-borne metastasis. We have previously shown[15] the close relationship between this classification and the five-year post-operative results. Here is another demonstration of its relation to prognosis.

TABLE VII

DISTRIBUTION OF CASES BY DUKES' CLASSIFICATION IN 32 CASES WITH VISCERAL METASTASES

PRESENT SERIES AND 1916-1932 SERIES

	A	B	C
Cases with visceral metastases selected from present and 1916–1932 series:			
Cases	0	8	24
Per cent	—	25%	75%
Present series (all cases)			
Cases	13	24	41
Per cent	17%	31%	52%
1916–1932 series:[15]			
Cases	20	49	40
Per cent	18%	45%	37%

Eight, or 25 per cent, of the 32 cases with visceral metastases were B-cases, and failed to show metastases in the regional nodes. Apparently blood-borne metastasis, before the nodes are involved, is not as rare as has been believed. In our 1916–1932 series the incidence of five-year

survivors after operation in the A-, B-, and C-cases was 100, 59, and 23 per cent, respectively. These results applied only to cases surviving operation five years and those dying of the disease. The fact that 100 per cent of the A-cases survived five years without disease, agrees with our findings that visceral metastases have not been found in the A-group. As for lymph node metastases, Dukes[14] and Grinnell[15] have shown that they almost never occur in the A-cases until the muscle layer of the bowel has been penetrated and the case can no longer be classed as an A-case. The difference between the survival rate of 100 and 59 per cent in the A- and B-cases can be largely explained by the greater liability of venous spread in B-cases, for as far as lymphatic spread is concerned all the A- and B-cases should have survived. It is possible, also, that some of the B-cases would have been classed as C-cases had the clearing technic been used in the search for node metastases. This would have raised the five-year survival rate of the B-cases somewhat nearer that of the A-cases.

TABLE VIII

FOLLOW-UP RESULTS ACCORDING TO DUKES' CLASSIFICATION IN 1916–1932 SERIES

Cases Surviving Operation Five Years and Those Dying of Cancer in That Period

		A	B	C
Cases	63	10	27	26
Five-year survivors:				
Cases	32	10	16	6
Per cent	51%	100%	59%	23%

When the cases with visceral metastases were studied for the presence of local vessel invasion, it was found to be present in nearly everyone. Out of the 30* specimens of rectal carcinoma removed at operation, vessel invasion was found microscopically in all but three, or 90 per cent. Brown and Warren were able to find it in all but one of their 70 cases with visceral metastases. In other words, the absence of vessel invasion after careful search in the sections from the primary growth will nearly always rule out the possibility of visceral metastases. Unfortunately, because of lack of sufficient follow-up period in our series, so far, the prognostic value of a positive finding of vessel invasion as regards visceral metastasis could not be determined. A negative finding, however, is probably far more important.

SUMMARY

(1) A series of 75 specimens of the rectum and rectosigmoid were cleared by the modified Spalteholtz technic, and studied for lymph node metastasis and blood vessel invasion.

(2) The average number of nodes per specimen was 52.

(3) Node metastases were found in 41 cases, or in 55 per cent. This incidence is 19 per cent higher than that found in a similar series of cases at this hospital, from 1916–1932, in which the clearing technic was not used.

(4) No intramural lymphatic spread of any significance was seen in any case.

* Two cases were omitted because sections were inadequate for vessel invasion study.

(5) The main extramural lymphatic spread is upward along the superior hemorrhoidal vessels. In over one-half of the cases with metastases, only three nodes or less were involved, and in over one-half of the cases the involved nodes lay within 3 cm. of the tumor. In nine, or 22 per cent, of the cases with metastases the most proximal nodes, near the point of ligation of the superior hemorrhoidal vessels, were involved. Thus, 40 per cent of the ten cases having perineal excision, and only 8 per cent of the 62 cases having abdominoperineal resection, fell into this unfavorable group, with a probably inadequate operation.

(6) In most cases with node metastases the upward spread was relatively progressive and orderly. In 17 per cent, however, it was definitely discontinuous.

(7) Only four, or 24 per cent, of the 17 cases with node metastases, having tumors below the peritoneal reflection, could have had adequate node removal by perineal excision.

(8) There was only one proven case of lateral lymphatic spread along the middle hemorrhoidal vessels, and only one instance of downward spread found in our series.

(9) Metastasis to the paracolic nodes was not found in any of the specimens. One case showed clinical evidence of extension along this route following operation.

(10) The incidence of node metastasis was 18 per cent higher when the tumor was completely annular than when it was not. No relationship could be demonstrated between the square area of the tumor and the frequency of metastasis.

(11) Blood vessel invasion was found in 36 per cent of 75 specimens of carcinoma of the rectum, and in 41 per cent of a combined group of 162 colon and rectal tumors.

(12) The incidence of blood vessel invasion was four times as great in the Grade III cases as in the Grade I. It was present in all the Grade III cases. Cases with visceral metastases showed a higher incidence in Grade III than unselected cases.

(13) The incidence of visceral metastasis and its relation to local blood vessel invasion could not be determined because of the insufficient follow-up period.

(14) There were no A-cases with visceral metastases. Eight, or 25 per cent, of the 32 cases with visceral metastases were B-cases, without node involvement.

(15) Twenty-seven, or 90 per cent, of the 30 cases with visceral metastases showed blood vessel invasion. Only three cases, or 10 per cent, failed to show it.

CONCLUSIONS

(1) The use of the modified Spalteholtz method for finding and charting lymph nodes will greatly increase the number of node metastases found, and will so aid in more accurate prognosis.

(2) The main extramural lymphatic spread is upward along the superior hemorrhoidal vessels. It tends to remain localized and to extend slowly in most cases. More distant spread, often discontinuous and unpredictable, is not uncommon, however.

(3) Lateral lymphatic spread along the middle hemorrhoidal vessels is probably infrequent and of secondary importance, occurring chiefly when the higher nodes are blocked by metastases.

(4) Downward lymphatic spread is exceedingly rare in operative specimens, and only occurs by retrograde flow when the high nodes are blocked.

(5) The rare occurrence of downward extension suggests that the operation of anterior abdominal resection, with ligation of the superior hemorrhoidal vessels, may have a wider field of usefulness for tumors near the rectosigmoid junction.

(6) Metastasis to the paracolic nodes is rare except in cases that are probably beyond operative cure.

(7) Perineal excision is a completely inadequate operation, even for carcinomata lying below the pelvic peritoneum.

(8) The tendency of rectal carcinoma to metastasize by way of the blood stream varies, in general, with the degree of differentiation of the tumor, and with the extent of local spread, as outlined by Dukes.

(9) Blood stream metastasis before the muscle wall of the rectum has been completely penetrated by the tumor is probably rare.

(10) Blood-borne metastasis after complete penetration of the muscle wall, but before the regional nodes are involved, is not as rare as has been believed.

(11) The value for prognosis of both the grading of tumors and their classification according to local spread has been further demonstrated.

(12) Failure to find local blood vessel invasion in the tumor after careful search is strong evidence that no visceral metastases exist.

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PRIMARY ABSCESS OF THE LIVER DUE TO ANAEROBIC NONHEMOLYTIC STREPTOCOCCUS

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ABSCESSES OF THE LIVER may be classified into two main groups: (a) Amoebic, which are usually single and which are associated with amoebic dysentery; and (b) pyogenic. The latter group may be due to invasion by a variety of organisms, and as noted by Ochsner,[6] may (1) follow infection in areas drained by the portal system, e.g., appendix, rectum and other portions of the bowel; (2) spread from contiguous structures, e.g., cholecystitis, gastric and duodenal ulceration, subphrenic space infection; (3) result from trauma by penetration from without or by infection in a hematoma with organisms already present in the liver; and (4) blood-borne infections *via* the hepatic arteries. In many instances no antecedent causal infection is demonstrable.

Although much has been written on the subject of pyogenic abscess of the liver, its bacteriology has not been emphasized. In a large series of collected cases, Ochsner[6] was able to find only 184 in which the bacteriology of the pus was reported. In these *B. coli*, streptococci and stayphylococci predominated. Failure to include the results of anaerobic cultures in many instances throws some doubt on the validity of the assumption that the above aerobes were the only organisms present. With the gastro-intestinal tract teeming with anaerobic bacteria, it does not seem as if they would be consistently absent from abscess of the liver. As future series of cases are reported, in which careful anaerobic as well as aerobic cultures are made, other organisms may be included as being etiologic. It is to be expected that anaerobes will be isolated more frequently.

We have had the opportunity recently to study a case where precise bacteriologic examination demonstrated the complete absence of the usual aerobic organisms isolated in abscess of the liver, and the exclusive presence of a strictly anaerobic nonhemolytic streptococcus. Three points in this case are of particular interest:

1. The presence of, and to our knowledge, for the first time recorded, a primary solitary hepatic abscess from which a pure culture of an anaerobic nonhemolytic streptococcus was recovered.
2. The unknown portal of entry of the organisms.
3. The inconstancy of the symptoms and clinical findings.

In 1893, Veillon,[10] described under the name of *M. foetidus,* a strictly anaerobic coccus, growing in short chains, and producing gas and a fetid odor in cultures, which he isolated from cases of Ludwig's angina, perinephric abscess and suppurative bartholinitis. In 1895, Kronig,[1] and Menge,[4] independently described a strictly anaerobic streptococcus which they found in the vagina in pregnancy, and later, in collaboration,[5] isolated several strains from the vagina and lochia in infected puerperal cases as well as from parametrial suppurations and peritonitis. Their organism was called *Streptococcus anaerobius.* Lewkowicz[2] (1901) isolated from the mouths of sucklings, a third variety of an extremely minute streptococcus growing only under strictly anaerobic conditions (*Streptococcus micros*).

The importance of these organisms in relationship to puerperal septicemia was first insisted on by Schottmuller[9] (1910), although their presence in the genital tract in the puerperium had been noted by several earlier workers.[8] Schottmuller's observations have been extended and confirmed by many other workers.[8] It would seem that the anaerobic streptococci form part of the flora of the normal genital tract.[8]

In recent years, numerous other reports have appeared in the literature describing the pathogenicity of anaerobic streptococci for man.[8] Dental infections harbor these organisms. They have been grown in pure or mixed cultures derived from chronic otitis media, cerebral abscess, meningitis and frontal sinusitis. Many believe that they are responsible for the severe infections following "human bites." Ulcerating carcinomata connected with the oral cavity frequently yield an aerobic streptococci on culture. They have been found in the blood in some cases of postanginal sepsis and bacterial endocarditis. They have been isolated repeatedly from abscesses of the lung and putrid empyemata. They have been cultured from the alimentary tract, including the gallbladder, and from exudates associated with appendicitis. Anaerobic streptococci in association with *B. coli* are said to cause the foul odor of peritonitis and appendiceal abscess, which is attributed popularly and erroneously to the colon bacillus alone. Bacterial synergistic gangrene of the abdominal or chest wall which sometimes follows operations on the gastrointestinal tract or pleural cavity has been found by Meleney[8] to be due to the symbiotic action of staphylococci and micro-aerophilic nonhemolytic streptococci. Urinary and genital tract infections due to anaerobic streptococci have been reported, and they have been recovered from traumatic wounds both civil and military.

As far as we can determine, only one case of abscess of the liver from which anaerobic streptococci were cultured is on record. McDonald, Henthorne and Thompson[3] reported a case of perforated duodenal ulcer, repaired surgically. Two and one-half months later, at necropsy, a huge subdiaphragmatic abscess was found, and also multiple chronic abscesses of the liver. The duodenal ulcer was entirely healed. Anaerobic nonhemolytic streptococci, in pure culture, were grown from the abscesses of the liver.

Case Report.—A. D., white, male, age 19, was admitted to Harkness Pavilion of the Columbia Presbyterian Medical Center, April 14, 1941, complaining of chills and fever of four weeks' duration. On March 19, 1941, two days after playing football in the snow, he felt chilly, and was admitted to the college infirmary. The following day he had a shaking chill and on subsequent days, had two other severe chills. There was persistent fever varying from 98.6° to 104.4° F., usually reaching a peak at 8 P. M. The patient awakened almost every day about 5:30 A. M. with a drenching sweat. He suffered from anorexia, with concomitant weight loss. During the first four weeks of his illness, he had a mild unproductive cough. About April 2, 1941, two weeks after the onset of the illness, he noticed a slight, inconstant pain in the right upper quadrant of the abdomen below the costal margin, which "caught" him when he breathed. This pain was present for two days and then subsided only to recur six days later. During the latter episode he complained also of constant pain in his right shoulder, made worse by deep inspiration. The shoulder was not tender nor was it hot or swollen. There was no history of diarrhea or of jaundice. There had been no known exposure to enteric disease. He had spent part of the preceding summer in Mexico, but otherwise had not been out of the United States. He had taken no unpasteurized milk. He had noted a small pustule on the back of the neck several months before the onset of his illness, which subsided without drainage. There was no history of familial disease.

The past history included (1) pneumonia in 1930, complicated by bilateral otitis media, (2) serum sickness in 1930, following tetanus antitoxin, and (3) uncomplicated measles in 1934. Operations included several partial tonsillectomies, the last in 1930, as well as myringotomies in 1930 and 1933. He had been exposed to tuberculosis in April, 1940, by a maid in the household who had contracted pulmonary tuberculosis. Physical examination at the time of college entrance in September, 1940, revealed a well-developed young man, weighing 165 pounds, with no pathologic findings other than slight dental caries. Dental roentgenograms in December, 1940, showed no abscesses.

The reports of the laboratory data which were brought in with the patient were as follows: Hemoglobin ranged from 12.2 gm. (84 per cent) to 13.5 gm. (93 per cent); R.B.C. from 4,010,000 to 4,270,000; the W.B.C. from 10,000 to 17,000; and the polymorphonuclears from 72 to 85 per cent. There was no eosinophilia, and there was a marked increase in the number of young polymorphonuclears. Urinalyses showed normal findings repeatedly. A single urine culture revealed *B. coli* and *Staphylococcus albus* Three blood cultures showed no growth. Five stool cultures yielded no enteric pathogens. Blood agglutination tests were negative for *B. typhosus*, *O* and *H* antigens; *B. paratyphosus*, *A*, *B*, and *C*; *B. abortus and melitensis*; *B. proteus X* 19; and for the Forssman antigen. Roentgenologic examinations of the chest, spine, and tibiae were negative.

No definite diagnosis had been made. Therapy had included courses of sulfathiazole, quinine bisulfate, and sodium salicylate. These were ineffective. Because no improvement took place the patient was transferred to the Harkness Pavilion, 26 days after the onset of the illness.

Physical examination, on admission, revealed a well-developed, but thin, chronically and acutely ill young white male, who showed obvious weight loss and pallor, and no jaundice or petechiae. Temperature 104° F., pulse 110, respirations 24, and blood pressure was 135/85. The right tympanic membrane was scarred. There were a few shotty posterior cervical lymph nodes. Examination of the thorax revealed inconstant tenderness over the 7th, 8th and 9th ribs on the right, and 9th, 10th and 11th ribs on the left. This tenderness was more marked on the right side. Examination of the lungs disclosed a short, inconstant friction rub over the 7th and 8th ribs anteriorly on the right side. At times, on deep inspiration, the patient grimaced with pain and at these times the right chest expanded less than the left. Abdominal examination showed right upper quadrant tenderness and slight spasm. However, there was no costovertebral angle tenderness, and the liver and spleen were not palpable. By percussion, the liver dullness

extended from the right 5th rib to the costal margin. An interesting observation on subsequent examinations was the production of pain in the right shoulder with deep inspiration with the patient supine. This phenomenon could not be reproduced with the patient in the sitting position.

Repetition of the blood counts, blood agglutination tests, urinalyses and stool examinations yielded essentially the same results as those made prior to admission. Three blood cultures showed no growth after incubation under aerobic, anaerobic and CO_2 conditions.

The blood chemistry findings are shown in Table I:

TABLE I

BLOOD CHEMISTRY DATA

	4/16/41 Admission	4/26/41 Preoper.	5/10/41 Postoper.	6/25/41 Before Discharge
Serum inorganic phosphorus:	4.7 mg. %	4.6 mg. %	..	4.2 mg. %
Serum phosphatase:	12.1 Bodansky U.	11.8 Bodansky U.	..	4.6 Bodansky U.
Serum bilirubin:	Very faint trace	0.8 mg. %	..	..
N. P. N.:	21 mg. %	38 mg. %	..	29 mg. %
Serum cholesterol:	95 mg. %	113 mg. %	155 mg. %	167 mg. %
Free:	..	43 mg. %	52 mg. %	..
Ester:	..	70 mg. %	103 mg. %	..
Serum protein:	6.7 mg. %	7.5 mg. %	..	7.3 mg. %
Albumin:	3.6 mg. %	3.7 mg. %	..	4.5 mg. %
Globulin:	1.1 mg. %	3.8 mg. %	..	2.8 mg. %
Euglobulin:	..	0.9 mg. %	..	..
Cephalin flocculation test:	..	..	±	..
Bromsulphthalein test for liver function:	..	..	..	5% retention of dye after 30 minutes.

Additional studies included a plain film of the abdomen, which showed normal kidney, ureter and bladder shadows, but an indistinct right psoas shadow; the liver shadow was not enlarged and its lower margin was normal. Six chest films and fluoroscopic examinations showed normal diaphragmatic contours. However, there was a diminished excursion of both domes of the diaphragm and the right dome was consistently less mobile than the left. There was evidence of thickening of the right diaphragmatic pleura and a small amount of free pleural fluid was noted at the right lung base, but this latter finding was not constant.

Course.—The patient was studied for 18 days, during which time he received two whole-blood transfusions. He suffered repeated chills of varying intensity and the spiking temperature, ranging from 100.2° to 105.4° F., continued, as did the weakness, anorexia, and sweats. An otolaryngologist was unable to demonstrate any focus of infection in the ears, nose, throat or paranasal sinuses.

Because of the chills and spiking temperature, the vague pains in the right lower chest and right upper abdomen, the continued prostration of the patient, the leukocytosis, the disturbed blood chemistry, and the fluoroscopic finding of a consistently diminished excursion of the right diaphragm, a clinical diagnosis was made of a purulent infection near the right diaphragm, probably intrahepatic. The signs of right pleural irritation were thought to be secondary. Solitary amoebic abscess of the liver was regarded as a possibility, and a course of emetine hydrochloride was given, but with no effect. Accordingly, surgical exploration of the right upper quadrant of the abdomen was deemed advisable.

Operation.—May 2, 1941 (19 days after admission, and 45 days after the onset of the disease): Exploratory celiotomy was performed through a right paracostal incision. On opening the abdomen it was found that the liver was much larger than had been appreciated preoperatively, and extended 4–5 cm. below the costal margin. On the superior surface of the right hepatic lobe, an area of induration, 9 cm. in its greatest diameter, was felt. The dome of this indurated area felt softer than the surrounding

liver. There were numerous delicate adhesions between the liver and the diaphragmatic peritoneum. There was no pus in the subphrenic space. There was no evidence of infection in the right perinephric region, in the right lumbar gutter, or in the subhepatic region. The appendix appeared normal. A diagnosis of a deeply situated abscess of the right lobe of the liver was made.

The right 9th rib was resected directly over the suspected area on the superior surface of the liver. The pleura was incised and immediately sutured to the diaphragm. The diaphragm was then incised exposing the liver. Two strips of iodoform gauze packing were placed between the diaphragmatic peritoneum and the surface of the liver. The transpleural wound was packed with vaselined gauze. The abdominal incision was closed without drainage. Forty-eight hours later the packing was removed from the transpleural wound and a small suction trochar was introduced into the soft spot in the liver. At a depth of 3.5 cm. from the surface, 60 cc. of yellow-brown pus was encountered. The tract was enlarged with the electrocoagulator, without bleeding. A soft rubber catheter drain was placed into the cavity, and the wound was packed.

Course.—A transfusion was given during the first operation and another four days after the second operation. Examination of the pus showed the presence of minute gram-positive cocci in chains, but no amoebae nor other parasites. No chemotherapy was carried out. The postoperative course was uneventful. The white blood cells fell progressively to 7,600, with 53 per cent polymorphonuclears. Hemoglobin and red blood cells remained at normal levels. From the first day after the second-stage operation the temperature remained below 101° F., and the temperature, pulse and respirations were normal from the 20th postoperative day. The abdominal wound healed *per primam.* There was profuse drainage from the abscess for 20 to 25 days, with gradual subsidence. The patient was allowed up on the 44th postoperative day, and was discharged on June 26, 1941, 54 days after operation, in excellent condition, weighing 159 pounds, or six pounds under his average weight. He has remained well. When seen six months later he weighed 175 pounds, and had been attending college for two months.

Bacteriology.—Gram stain of the exudate showed the presence of many pus cells and a small number of minute gram-positive cocci in chains. Repeated examinations of fresh specimens failed to reveal the presence of amoebae, giardia, or other parasites. All dark field examinations were negative. Aerobic 5 per cent sheep's blood agar plates gave no growth. Anaerobically however, there was growth in dextrose meat broth and on the blood agar plates. Smears from the broth showed the same gram-positive cocci seen on direct smear of the exudate. The blood agar plates yielded a pure culture of the same organism. The biochemical reactions of this organism corresponded to those of the *Streptococcus intermedius,* of Prevot.[7] The organisms remained strict anaerobes on repeated subculture. Subsequent cultures of the pus also grew out this anaerobic nonhemolytic streptococcus.

SUMMARY

1. This is the first report, as far as the authors have been able to determine, of a primary, solitary abscess of the liver from which a pure culture of an anaerobic nonhemolytic streptococcus was recovered. Careful examinations revealed no apparent portal of entry of the organisms.

2. A brief review of the literature on the pathogenesis of the anaerobic nonhemolytic streptococci is presented.

3. The inconstancy of the symptoms and localizing signs in this case are significant.

4. Pain in the shoulder with deep inspiration in the supine position and its absence in the sitting position are offered as a contributory sign in the diagnosis of abscess of the liver.

5. The importance of routine anaerobic as well as aerobic cultures is emphasized by this case.

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SEVERE TRAUMA TO THE LIVER WITH "HEPATORENAL SYNDROME"

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THE CLINICAL IMPORTANCE of hepatic failure as a cause of collapse in certain cases following operation upon the biliary tract was first emphasized by Heyd (1924). Since then a number of clinical and experimental articles on "liver death" and "liver-kidney failure" have appeared. The papers of Boyce, Helwig, and associates, and Wilensky, are particularly valuable for their discussions of this somewhat controversial subject. Furtwaengler (1927) is said to have been the first observer to report a case in which the possible relation between trauma to the liver and the subsequent development of damage to the kidney was recognized. Recently, Orr and Helwig (1939) directed attention to the importance of the recognition of the "hepatorenal syndrome" in certain cases of severe trauma to the liver. Boyce's monograph (1941) offers an excellent review of the subject of hepatic insufficiency.

The following case demonstrates some interesting clinical, physiologic, and biochemical observations made on a healthy young adult patient who received an injury limited to the liver:

Case Report.—A male, age 20, was injured in an automobile accident, September 3, 1940. The lower thoracic and upper abdominal regions were crushed between the engine and the front seat of the car. Fifteen minutes after the accident he was brought to the hospital in profound shock—pale, cold, and restless—but conscious and complaining of abdominal pain. Temperature 98° F., pulse 120, blood pressure 40/20. There were lacerations and abrasions about the face and mouth. Generalized guarding and tenderness were present throughout the abdomen, most marked in the right upper quadrant. Peristaltic activity was diminished. Treatment for shock included an intravenous infusion of 1000 cc. of glucose solution, and a transfusion of 500 cc. of citrated whole blood. After six hours of observation his condition had improved and the blood pressure was 114/70. The abdominal pain and signs of peritoneal irritation had increased. The pulse rate remained about 128, but was considerably improved in quality. At that time, the hemoglobin content of the blood was 120 per cent, the red blood cell count 5.65 million, and the white count 53,000. About six hours after the patient had sustained the injury, abdominal exploration was undertaken under ether anesthesia. Another transfusion of citrated whole blood was given during the operation. A deep laceration, about 10 cm. in length, was felt in the dome of the right lobe of the liver. It was bleeding freely and the abdomen was filled with blood; 1500 cc. of blood was aspirated from the abdominal cavity, but autotransfusion was not performed as the blood contained bile. The laceration in the liver was inaccessible for suturing, and a large, hot, moist pack was inserted into the defect, while the remainder of the abdomen was explored, but no further injury was found. The pack was removed and the bleeding

had ceased. A Penrose drain was placed to the laceration in the liver and brought out through a small separate wound in the right lateral subcostal region. The abdomen was then closed. The operation lasted 53 minutes and was tolerated fairly well. The patient reacted from his anesthetic in the usual length of time. His pulse and blood pressure were within normal limits for several hours, and during this early postoperative period he voided a total of 240 cc. of normal urine. Shortly after the operation, the temperature was 103.4° F., and it remained at about this level until the next day. On the morning after the operation the patient appeared weak, the pulse was very rapid and of poor volume (uncountable), and the blood pressure had fallen to 88/55. That afternoon he became wildly delirious and the temperature rose to a maximum reading of 107.8° F. rectally; the extremities were cold and cyanotic, the pulse was barely discernible, and the systolic blood pressure varied from 70 to 80. The abdomen remained flat, soft, and without tenderness. There was no urinary output for 19 hours, and catheterization yielded only 50 cc. of dark urine containing a trace of albumin and an occasional cast. The nonprotein nitrogen in the blood was elevated to 110 mg. per 100 cc. of blood. Parenteral fluids, glucose and physiologic saline were given and continuous oxygen inhalation therapy was provided. Three transfusions, each consisting of 500 cc. of citrated whole blood, were given over a period of several hours. Sedation was accomplished with paraldehyde. Following the first of these transfusions the blood pressure was increased to 100/80, and the urinary output gradually began to increase. The temperature declined to 103° F., and remained around this level for the subsequent three days and then gradually became normal. He was comatose and restless during the first two days of the reaction and then rather suddenly became mentally clear. The abdomen remained soft and nontender throughout the entire postoperative period. There was no further hypotension or oliguria and the azotemia decreased rapidly. A few red blood cells appeared in the urine on the second day after operation and such cells were found thereafter for several days. Albuminuria was present for six or seven days and then disappeared. Tests of the liver and kidney function by excretion of dye were not performed until the fifth postoperative day and at that time they were within normal limits. Mild clinical icterus developed and the icteric index was 38 units on the third day following the injury, 21 on the fourth postoperative day and 12 on the fifth day. The prothrombin time was estimated as 80 per cent of normal on the third day and 100 per cent on the fifth. During the first day a considerable amount of bile-tinged serous fluid soaked the dressings covering the drainage wound, and a moderate amount of clear bile drained for several days thereafter. There was no evidence of bleeding from the drainage wound at any time after the operation. After a stormy course during the first four days, the patient made a good recovery. He was discharged from the hospital, October 7, 1940, and has remained well. Chart I and Table I show the more important laboratory determinations that were made during the critical part of his illness. The severity of the illness, and the urgent need for active treatment necessarily limited the laboratory studies.*

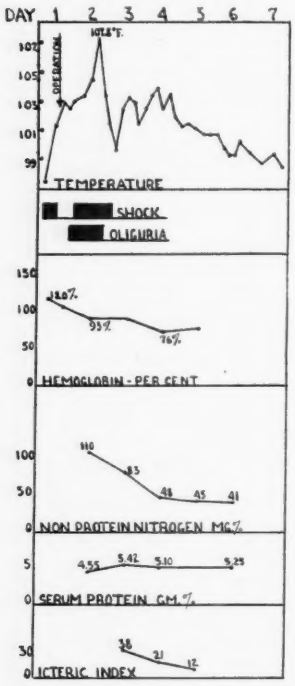

CHART I

* The biochemical determinations were done by Mrs. Ruth Nelson Cornell in the laboratory of Dr. Raymond Reitzel.

TABLE I

LABORATORY DATA IN THE CASE OF A PATIENT WITH SEVERE LIVER TRAUMA WHO DEVELOPED COLLAPSE, WITH VERY HIGH TEMPERATURE AND OLIGURIA ON THE DAY FOLLOWING HIS INJURY. THE LIST INCLUDES THOSE EXAMINATIONS THAT WERE MADE DURING THE FIRST SIX DAYS.

Blood: Date	9-3-40	9-4-40	9-5-40	9-6-40	9-7-40	9-8-40
Hemoglobin (14.5 g = 100%)	120%	93%	93%	76%	79%	—
Erythrocytes (millions per cu. mm.)	5.65	—	4.30	3.90	4.60	—
Leukocytes (number per cu. mm.)	53000	19000	14000	10000	19000	—
Urine:						
Specific gravity	QNS	1.022	1.027	1.028	1.026	—
Albumin		Trace	Trace	Trace	Trace	—
Casts	—	Few	Few	Few	Few	—
Red blood cells (per h.p.f.)	0	0	2	5	50	10
Urobilin (Schlessinger's test)	—	—	0, 1:10	+, 1:1	+, 1:40	—
Bile	—	0	0	0	0	—
Blood Chemistry:						
Nonprotein nitrogen (mg. %)	—	110	83	48	45	41
Creatinine (mg. %)	—	—	2.6	—	2.9	—
Plasma chlorides (mg. %)	—	663	679	—	—	623
Serum protein (Gm. %)	—	4.55	5.42	5.10	—	5.25
Serum albumin (Gm. %)	—	3.56	4.36	4.20	—	3.62
Serum globulin (Gm. %)	—	0.99	1.06	0.90	—	1.63
Icterus index (units)	—	—	38	—	21	12
Glucose (mg. %)	—	—	112	—	—	—
Plasma cholesterol (mg. %)	—	—	159	—	—	—
Prothrombin time (H. P. Smith method)	—	—	—	80%	—	100%
Rose bengal (retention in serum) 8 minutes	—	—	—	—	—	63%
(liver function test) 16 minutes	—	—	—	—	—	43%
Phenolsulfonphthalein excretion (2 hours) (kidney function)	—	—	—	—	—	65%
Fluid intake (water, glucose & saline — cc.)	2000	2000	3000	4800	4600	3600
Transfusions (citrated whole blood — cc.)	1000	1500	850*	—	—	—
Urinary output (cc.)	670	700	1250	1270	1880	2170

* Plasma.

The clinical and pathologic changes that occur in the "hepatorenal syndrome" have been collected and described by Boyce, and by Orr and Helwig. These changes are most likely to follow extensive crushing or gunshot injuries of the liver rather than stab or incised wounds. In some of these cases of severe trauma to the liver, hyperpyrexia and alterations in the blood chemistry follow the injury in a few hours. The rise in temperature is often very high and is associated with a rapid, weak pulse. Restlessness, collapse and delirium may accompany the high febrile reaction.

The alterations in the blood chemistry often appear early and usually consist of an increase in the nonprotein nitrogen and creatinine. Icterus may occur. The blood glucose carbon dioxide combining power, cholesterol, and plasma chloride levels have not been altered in the few cases in which they have been determined.

Alterations in renal function are likely to occur also and may appear early, as in our case, or be delayed. Oliguria, or anuria, and the appearance of red blood cells, white blood cells, casts and albumin in the urine are the usual findings.

In the case reported the elevation in hemoglobin and number of erythrocytes (120 per cent hemoglobin, 5.65 million red cells) on the day of the injury was probably the result of hemoconcentration that accompanies shock.

There was no evidence of such concentration during the period of collapse which occurred the day following operation when the extreme hyperpyrexia was present. The diminution in the level of plasma protein in the blood (4.55 Gm. per 100 cc. of blood) on the day following the injury most likely resulted from loss of plasma from the blood stream by reason of shock and hemorrhage. The plasma protein levels might well have been lower had not transfusions of blood and plasma been given.

The critical condition of patients with severe injury to the liver, and the urgent necessity for active therapy, have made it difficult or impossible, as in the case described above, to perform tests of liver function early in the course of the illness. The pronounced oliguria rendered the simple intravenous hippuric acid test impossible of performance, and the necessity for multiple venipunctures almost prohibits the use of the dye excretion or serum bilirubin tests while active therapy is in progress. Perhaps future cases of less severe trauma to the liver will permit more adequate examination for disturbances of liver function by serial Quick (hippuric acid) tests. The rose bengal excretion test made on the sixth day (Table I) on the case herewith reported, indicates remarkably good hepatic excretory function at that time, and the phenolsulfonphthalein test done on the same day shows evidence of good recovery of renal function. The prothrombin time of the blood was 80 per cent of normal on the third day following the injury and 100 per cent on the fifth day. These determinations were made relatively late, after numerous transfusions of blood had been given.

The pathologic changes in the livers of patients who have died of the traumatic "hepatorenal syndrome" are said to be degenerative, consisting of edema and necrosis, and to be confined principally to the cellular portions of the traumatized region. Diffuse, degenerative renal lesions have also been described and are usually greatest in the convoluted tubules.

Orr and Helwig attempted to explain the "hepatorenal syndrome" following trauma to the liver by suggesting the possibility that a soluble toxin is produced as a result of necrosis of the liver and that this toxin may produce injury to the kidney. They commented, however, that the "explanation of the causative factor or factors and the mechanism of their action upon the liver and kidneys must be determined by future investigation."

Apparently, Boyce also believes that a toxin is elaborated as a result of the degeneration of injured liver tissue (liver autolysis) but feels that the renal injury may be due to an "increase of its normal detoxifying duties, which are increased by failure of the detoxifying function of the liver, rather than by any specific action of the toxin."

Coller, in his discussion of Orr and Helwig's paper (1939), suggested that the azotemia and oliguria might be explained entirely on a basis of such alterations in physiologic processes as shock, dehydration, alkalosis, hypochloremia, hyponatremia, etc., rather than by the action of a specific toxin generated in the damaged liver. Other authors have suggested the possible

rôle of anaphylaxis and of infection, in attempting to explain this condition.

The sequence of events and the findings in our case appear to justify its designation as an example of the so-called "hepatorenal syndrome" following hepatic trauma. The patient was a young healthy adult. He sustained an injury in which the trauma apparently involved the liver exclusively. This injury was accompanied by intra-abdominal hemorrhage and profound shock. After a favorable response to therapy for shock, celiotomy was performed under ether anesthesia. There was profuse bleeding from a long deep laceration in an inaccessible part of the right lobe of the liver, which was controlled by the temporary application of a hot moist pack. Following the operation the patient reacted well at first, although the temperature remained around 103° F. About 30 hours after the injury an unusual clinical picture developed characterized by hyperpyrexia (highest 107.8° F.), rapid pulse of poor volume, hypotension (systolic pressure from 70 to 80), oliguria, retention of nitrogen, cold, moist, cyanotic skin and delirium with great restlessness evidenced by wild thrashing movements. Therapy consisted of continuous inhalations of oxygen and infusions of whole blood, plasma, glucose and physiologic saline solutions. After a stormy convalescence of several days the patient recovered completely. There was no evidence of infection, and nothing to suggest anaphylactoid or transfusion reactions.

Speculation regarding the reaction that occurred in this case brings up the possibility that shock with hypoproteinemia, diminished blood volume with decreased circulatory efficiency and hypoxia in the body tissues, including those of the liver and kidneys, may well have played a contributory part in the syndrome that followed. The trauma of the exploratory operation, the application of the hot pack to the damaged liver, and the use of ether as an anesthetic agent must also be considered as possible contributory factors in this case. Hypochloremia was not present; nor did Orr and Helwig find it in their cases. We feel that dehydration probably did not play a part in this case, although the fluid intake during the first two 24-hour periods was not high (3000 and 3500 cc., respectively, including the blood transfusions). Infection apparently played no part. At no time after operation were there signs of peritonitis, infection in the wound or infection in the bile which drained. Anaphylaxis and transfusion "reaction" were considered but thought to be very unlikely.

The possibility that some other factor (or factors) may be responsible for the production of this sudden, violent "hepatorenal syndrome" which sometimes follows severe trauma to the liver must also be considered. It may finally prove to be some series of physiologic and biochemical alterations, as suggested by the discussion of Coller. Or perhaps it is the soluble nephrospecific toxin originating in the liver, as suggested by Orr and Helwig, or a toxic product of autolysis of the liver, as postulated by Boyce. At present the final explanation is not evident.

Whatever the chief cause of the "hepatorenal" reaction may eventually prove to be, at the present time the prevention and treatment of this syndrome would appear to be best directed toward the correction of those aspects of the physiologic and biochemical alterations that are now known, even though they may prove to be only contributory factors. The active treatment of shock with transfusions of blood or plasma given repeatedly and in large amounts if necessary to replace lost blood, restore the blood protein level and thereby maintain adequate blood volume and circulatory efficiency, is of primary importance. Transfusions also provide protein and prothrombin in readily available form if deficiencies develop. An atmosphere rich in oxygen should be provided in order to insure, if possible, adequate oxygenation of the tissues, including those of the liver and kidney. The intake of fluid must be carefully and correctly determined; enough but no great excess of crystalloid solutions is provided. Hypochloremia has not been a factor in these cases. The requirements for sodium chloride solutions are, therefore, only for maintenance unless, for some other reason, vomiting for example, deficiency should develop. "Glucose is the sheet-anchor of therapy in patients with hepatic and biliary tract disease." (Boyce). This probably holds true for acute trauma to the liver as well as other conditions affecting its function, and glucose should, therefore, be provided in reasonably large amounts. Every infusion of fluid (except the blood transfusions) given intravenously to the patient with this type of trauma should probably contain glucose; physiologic saline is included in some of the infusions in amounts sufficient to provide for the calculated need for sodium chloride. Since there is some indication that dehydrocholic acid (Decholin) therapy may improve function of the liver when it is depressed (Boyce), and probably that this therapy causes improved function of the kidneys through improved hepatic function, it would appear reasonable to try it in these cases of severe injury to the liver. It was not used in the authors' case.

The operation to be performed in cases of severe trauma to the liver has been adequately discussed elsewhere. Many factors determine the decision as to whether or not to operate, the selection of the proper time for operation, and the procedure to be carried out. The importance of shock and hemorrhage, the type and probable extent of the injury to the liver, and the possibility of other internal injuries must be carefully considered in each case. Ordinarily we should treat shock and then operate as soon as a reasonably favorable response is obtained. Occasionally, however, evidence of continuing internal hemorrhage may necessitate operation after therapy for shock has been followed by little or no improvement in the patient's condition. In this connection the use of transfusions of blood before, during and after operation may be of considerable assistance. Oxygen therapy is also employed, and the anesthetic agent should be selected and administered with the idea of adequate oxygenation in mind. It is probably superfluous to advise that the least possible trauma be inflicted upon the patient, and especially the

already damaged liver, during the course of the operative manipulations. The question of whether to suture the liver, pack it, leave it alone, or possibly resect the damaged portion (as suggested by Tinker, and Boyce) must be decided in each case, depending on the condition of the patient and the location and extent of the injury. In some cases it may be possible and advisable to suture lacerations in the liver. Packing should be avoided, if possible, as it almost always leads to infection, or secondary bleeding, if it is left in place for any considerable length of time. In cases in which the necrosis is extreme, Boyce advocated resection of the traumatized hepatic tissue as an attempt to prevent "intoxication" from "liver autolysis." He mentioned the probable value of the thrombin spray of Warner as an effective means of hemostasis during resection of the liver. His thesis is interesting and the procedure of resection of traumatized liver may become a reasonable one, if the idea proves to be as true as it appears to be from his experimental and clinical observations. The critical state of the patient from shock and hemorrhage, and the extent and location of the injury to the liver are factors that must be considered, in addition to that of hemostasis in the liver, before resection of traumatized liver is undertaken. The duration of the operation is also a matter that may influence the outcome in a case of severe injury to the liver. Even though the surgeon is trained in the niceties of the Halsted method of surgical procedure in which gentleness, deliberation and thoroughness become habitual through many years of training, he may be inclined to agree with Boyce, who also cited Prey and Foster, that the operation in cases of severe trauma to the liver should proceed as expeditiously as is reasonably possible, mainly in order that the patient be spared the effects of prolonged anesthesia. Thoroughness, gentleness, and hemostasis, however, should not be sacrificed for the sake of shortening the operating time, because shock may be precipitated or increased by rough handling and extra bleeding, and dangerous injuries to other viscera may go undiscovered in perfunctory exploration of the abdomen.

SUMMARY

A case of severe trauma to the liver accompanied by shock and hemorrhage is reported. After favorable response to therapy for shock and operation for control of hemorrhage the patient responded well for several hours. The following day, about 30 hours after injury, he developed extreme hyperpyrexia, azotemia, oliguria, collapse, with cold, moist, cyanotic skin, hypotension, rapid thready pulse and delirium. The main part of his treatment consisted of transfusions of blood and plasma, continuous inhalations of oxygen and parenteral glucose therapy. The patient recovered.

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PLATYBASIA WITH INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM

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PLATYBASIA (synonyms—basilar impression, basilar invagination) is a deformity of the occipital bone and the upper cervical vertebrae. The nature and degree of the deformity varies greatly but, essentially, the foramen magnum is displaced upward into the cranial cavity and the upper cervical vertebrae are dislocated upward and forward into the cranial depression. In the majority of cases the deformity is due to a congenital defect in development of the occipital bone, atlas and axis, but in a smaller number the deformity is acquired as a result of some pathologic process that occurs in the bones of the region.

In platybasia the relative location, size and shape of the foramen magnum as well as that of the upper cervical canal is altered, and the posterior cranial fossa is diminished in capacity. In some this may not produce untoward symptoms at any time in life. In others, it may be associated either with congenital anomalies of the nervous system or be the direct cause of progressive nervous changes; sometimes both conditions exist. The resultant clinical manifestations may be confused, principally, with those occurring in conjunction with neoplasms of the posterior fossa or upper cervical canal, hydrocephalus, syringomyelia, syringobulbia, multiple sclerosis and spinocerebellar disease.

The four cases reported here will serve to demonstrate various etiologic factors, the structural changes and the nervous manifestations associated with platybasia. The results of operative decompression indicate the value of operation in alleviating symptoms and in staying the progress of the changes in the nervous system.†

Case 1.—*Platybasia and congenital deformity of the upper three cervical vertebrae. Neurologic signs interpreted as multiple sclerosis appeared at age 31, and progressed to marked disability. Operative decompression of the cerebellum and upper cord was followed by considerable improvement and partial rehabilitation after two years.*

H. DeS., white male, age 37, was admitted to the New York Hospital, January 23, 1940, complaining of weakness of all extremities, progressing for six years, almost to complete incapacitation.

In 1934, he first noticed weakness and clumsiness in the right upper extremity. Prior to this time his history had been unremarkable. Later in the same year his gait became staggering and he had sensations "akin to numbness" in his right hand and toes.

† Only the first three cases were presented at the meeting of the New York Surgical Society.

He was first examined in this hospital in October, 1936, when he was found to have coarse horizontal nystagmus, loss of fine motions, and weakness in the right hand, impaired discriminatory sense in the right hand, sluggish abdominal reflexes, unsteady gait, inability to stand with feet together, unsustained ankle clonus bilaterally, and a positive toe-stretch and Chaddock sign bilaterally. Cerobrospinal fluid findings were normal. A tentative diagnosis of multiple sclerosis was made. The same diagnosis was made in two other clinics during the ensuing four years, and he became steadily worse under various forms of treatment.

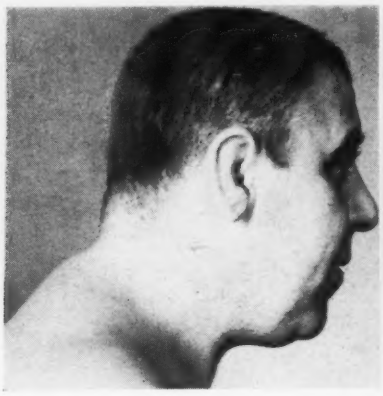


FIG. 1.—Case 1: Photograph showing a male, age 37, with congenital platybasia.

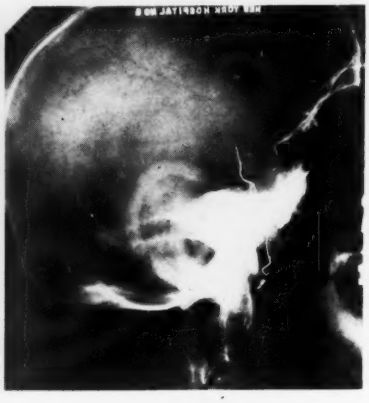


FIG. 2.—Case 1: Lateral roentgenogram showing rudimentary development of the occipito-atlanto-axial region, "occipitalization" of the atlas, upward dislocation of the odontoid process, and fusion of the second and third vertebrae.

In 1940, when readmitted to this hospital, the weakness and clumsiness in the extremities had progressed to the point where he was totally unable to dress himself, and even had great difficulty in feeding himself. He walked with a broad, staggering gait, requiring the partial support of another person. Vision often became blurred, particularly if he looked downward or sidewise. Vertigo occurred regularly on changes in position of the head. He also found that movement of the head caused a dull aching pain in the neck and back of the head.

Physical Examination.—The patient was heavily built and tended to be obese. The neck was short and thick and the head appeared to be thrust forward (Fig. 1). Speech was slightly dysarthric. There was coarse nystagmus on moving the eyes in any direction. The tongue deviated to the left. Other functions of the head and neck were normal.

In the upper extremities there was weakness in the hand grips, more pronounced on the right. Rapid rhythmic movements were badly performed. Point to point tests were grossly dysmetric. The deep reflexes were very active and there was a positive finger-stretch reflex bilaterally. Two-point discrimination, identification of objects by feel, and position sense were absent in both hands. Vibratory sense was diminished. Other forms of sensation were normal.

Sensation over the trunk was normal. The abdominal reflexes were sluggish and quickly exhausted.

In the lower extremities alternating movements and point-to-point tests were poorly performed. Gait was slow, staggering and broad based. Standing with feet together was impossible. The knee and ankle jerks on the left were exaggerated. Plantar responses were abnormal bilaterally. There was marked impairment of position and

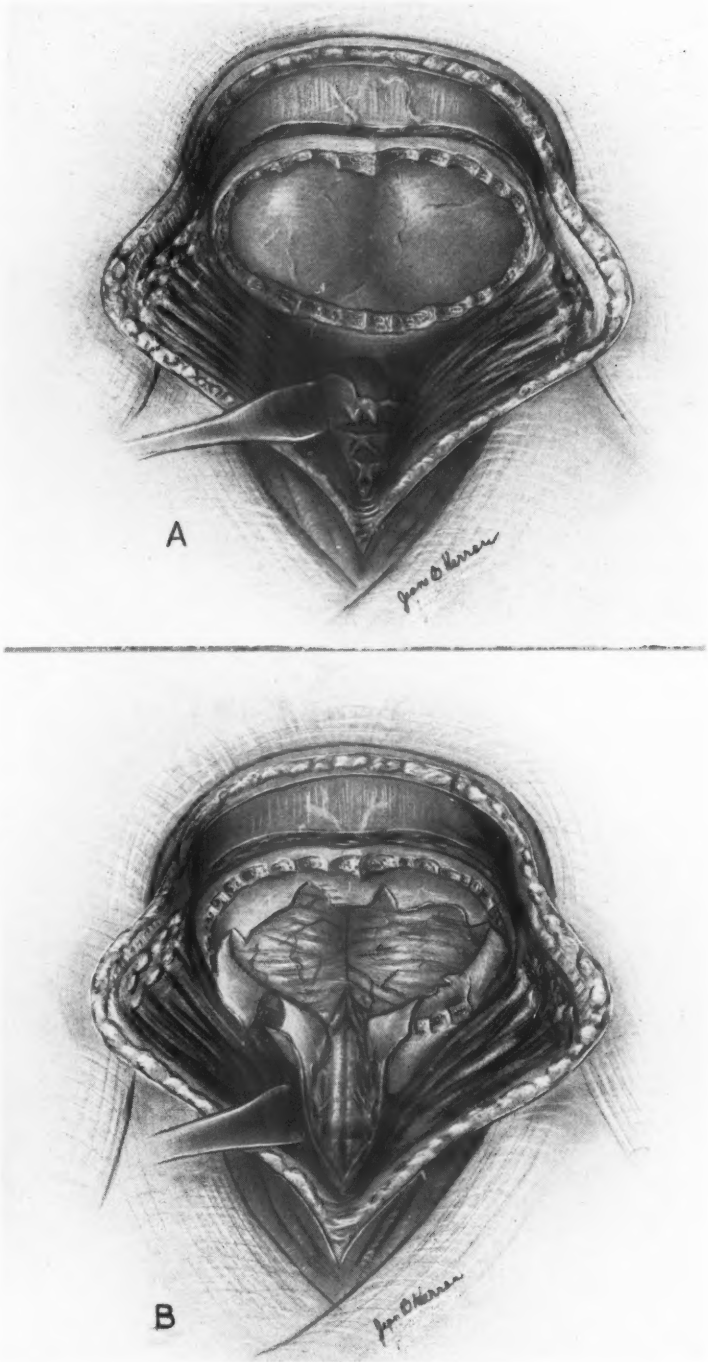


FIG. 3.—Case 1: (A) View with dura over cerebellum partly exposed showing thickness of the occipital bone at the foramen magnum due to "occipitalization" of the atlas. The arch of the axis is asymmetrical and its lumen narrowed. (B) View after opening the dura showing flattening of the cerebellum, narrowing in region of the foramen magnum, atrophic appearance of the upper cord, and transverse groove across cord at the level of the arch of the axis.

vibratory sense and moderate impairment of deep pain sense; other sensations were normal.

Roentgenograms (Fig. 2) showed absence of a discernible first cervical vertebra, due probably to its partial fusion with the occipital bone; thickening of the posterior margin of the foramen magnum; moderate protrusion of an abnormally long-appearing odontoid process into the foramen magnum; partial fusion of the laminae and bodies of the second and third cervical vertebrae.

Operation.—January 27, 1940: (Fig. 3). Local procaine anesthesia was employed; and a suboccipital and upper cervical decompression was performed. The attachment of the occipital muscles was low on the skull and exposure of the occipital bone was difficult. The foramen magnum appeared unusually small, and the bone about it unusually heavy. A flange of bone existed on the right margin of the foramen magnum, the result of fusion of the lamina of the first vertebra. The arch formed by the laminae of the second cervical vertebra appeared to be unusually narrow and to constrict the dura. After removal of a fairly large segment of the occipital bone, the foramen magnum was opened widely and the first two (second and third) cervical arches were also removed. The dura, particularly at the level of the foramen magnum, was abnormally thick (2 to 3 mm.) and tended to constrict the structures within it. After the dura was opened over the cerebellum an upper cord examination showed the flattened vermis and hemispheres of the cerebellum. The cisterna magna did not exist. The cerebellar tonsils were small and atrophic. The upper part of the exposed cord was void of its normal configuration and the pia arachnoid was adherent. At the level of the first intact vertebral arch there was a transverse groove in the dorsal surface of the cord that had a brownish pigmentation as though resulting from pressure of the neural arch. Below this point the cord appeared normal. The wound was closed but the dura was left open for decompression.

Postoperative Course.—Recovery from operation was uneventful and three weeks after operation the patient was thought to be improved, in that there was less discomfort in the neck, the "feeling of heaviness" in the upper extremities was less, position sense in the upper extremities was improved, and there was greater steadiness in standing and walking.

Follow-Up.—In the two years following operation improvement was gratifying. He was able to dress himself, walk about without assistance, and work with tools as a handy man. He was free of discomfort in the neck and of vertigo. Examination showed that none of the motor or sensory changes observed prior to operation had disappeared but all were improved.

COMMENT.—The steady progression of the neurologic symptoms during the six years the patient was under observation was striking. The rôle of the craniovertebral compression of nervous structures as a cause of the symptoms and the value of decompression, are incontrovertible in view of the marked improvement that followed operation. There is no reason to doubt that the skeletal anomaly was congenital but just why symptoms did not appear before age 30 is not obvious.

Case 2.—*Platybasia and congenital deformity of cervical spine. Neurologic signs simulating syringomyelia were slowly progressive for 16 years. Operative decompression of the cerebellum and upper cord was followed by improvement.*

G. J., white, male, age 43, was admitted to the New York Hospital, July 7, 1941, complaining of impaired sensation and recurring infections in the fingers of the left hand.

During childhood he had severe rickets, as did several other children in his large family. Otherwise his health was not noteworthy until 1925 (at age 27), when he developed painless infections in two fingers of the left hand and the last phalanx of each was amputated. The ends of two other fingers of this hand were lost under similar

circumstances during later years. Because of an infection in the left thumb he was first examined in this hospital in 1939, when he was found to have deformity and muscular atrophy of the neck, possibly slight wasting in the muscles of the left hand, absent tendon reflexes in the upper extremities, and impaired pain and temperature sense in the neck and upper extremities, more pronounced on the left. *Clinical Diagnosis:* Syringomyelia.

When admitted to the hospital in 1941 he added to his previous complaints those of more recent periods of vertigo, drooping of the left shoulder, increase in his neck-size and unsteadiness when working on scaffoldings.

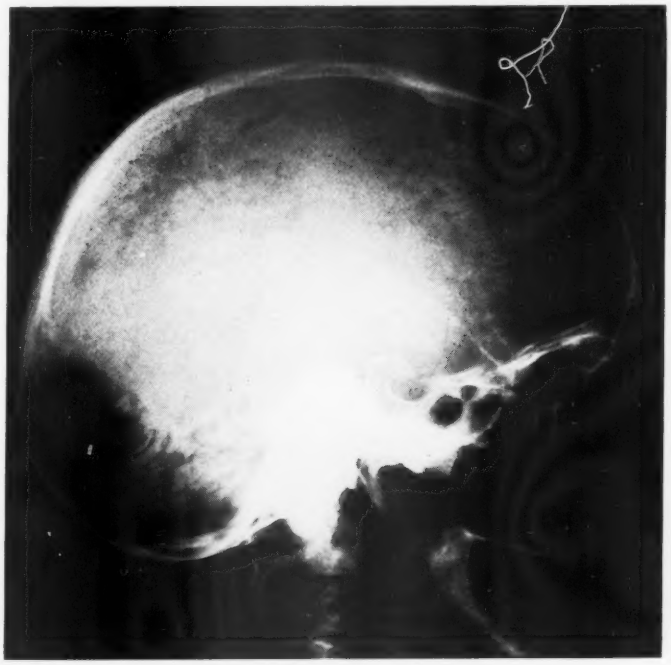


FIG. 4.—Case 2: Lateral roentgenogram showing long, narrow shape of the cranial cavity; prominence of the petrous ridges; upward displacement of the clivus blumenbachii; inclusion of the arch of the atlas in the foramen magnum; and upward displacement of the odontoid process.

Physical Examination.—The patient was of short, muscular build. The neck was short, thick and asymmetrical. The head was large in its biparietal measurement, and was held with the chin tilted to the left. There was slight limitation of motion in turning and extending the head on the neck. The left shoulder drooped, the angle between it and the neck was obliterated and the left sternocleidomastoid muscle was weak. There was a long S-shaped scoliosis involving the cervicodorsal spine. There was slight wasting as well as fibrillations of muscles in the neck and shoulder girdle on the left.

There was coarse nystagmus on lateral movement of the eyes, more pronounced on looking to the left. The protruded tongue deviated to the left and speech was slightly dysarthric. There was impairment of pain and temperature sense over the posterior part of the head, neck and shoulder on the left. Other functions of the head and neck were normal.

In the upper extremities the only demonstrable palsy was slight wasting of the muscles of the ulnar side of the left hand. The tendon reflexes were absent bilaterally. There was partial to total loss of pain and temperature sense in the left extremity and slight impairment of these sensations in the right. Other forms of sensation were pre-

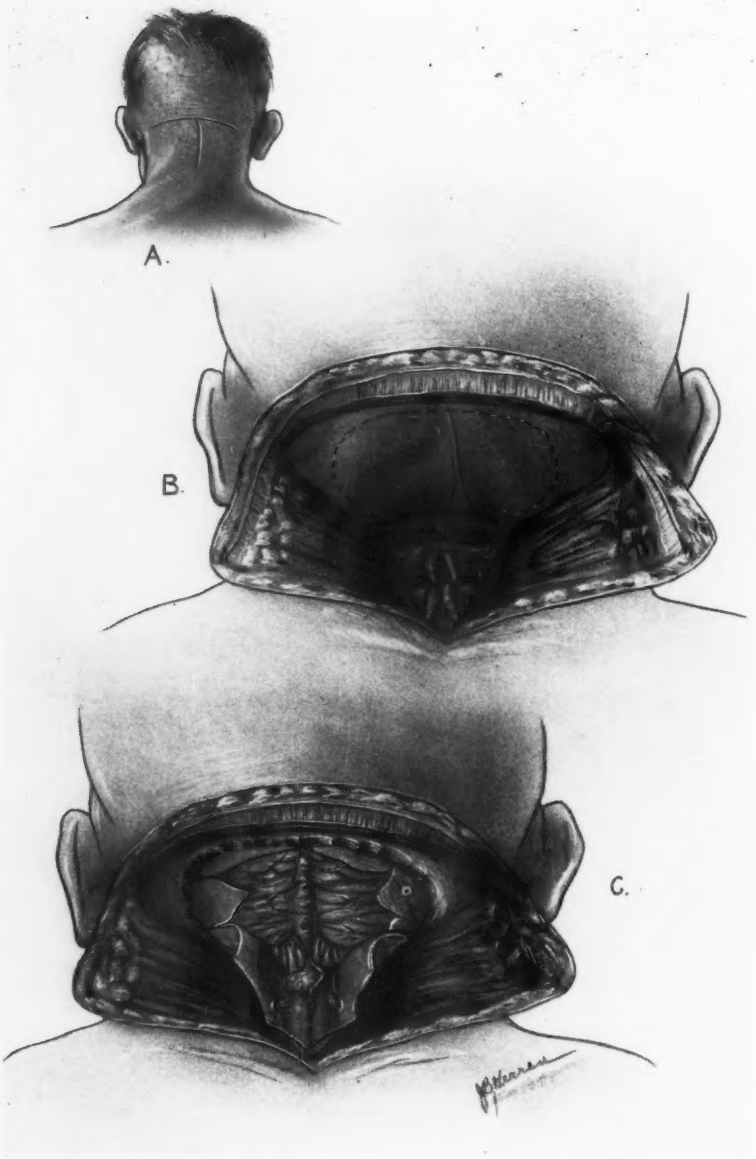


FIG. 5.—Case 2: (A) Type of operative incision. (B) View with occipital bone and upper three cervical vertebrae exposed, showing asymmetry and concavity of occipital bone, and partial inclusion of the arch of the atlas in the foramen magnum. (C) View after opening the dura showing compressed appearance of the cerebellum; herniation of the cerebellar tonsils; narrowing of the foramen magnum; an aberrant artery; and bulging at the junction of the medulla and cord.

served. The distal phalanges of all but the thumb on the left were absent while the thumb was swollen, deformed and ulcerated.

Sensation over the trunk was normal and the abdominal reflexes were absent. In the lower extremities the findings were normal but for a positive Romberg sign and impairment of position sense in the feet.

The cerebrospinal fluid pressure, manometric studies and laboratory tests were normal.

Roentgenograms (Fig. 4) showed partial fusion of the bodies of the fifth and sixth cervical vertebrae, protrusion of the odontoid process into the foramen magnum, partial inclusion of the neural arch of the atlas inside the foramen magnum, unequal prominence of the petrous ridges, and upward displacement of the clivus blumenbachii.

Operation.—August 8, 1941. (Fig. 5). Under ether anesthesia, a suboccipital and upper cervical decompression was performed. The skull between the inion and the foramen magnum was flattened and slightly concave. The middle ridge of bone was displaced to the right indicating an asymmetry of the posterior fossa. The neural arch of the atlas was almost hidden inside the foramen magnum. A segment of the occipital bone was removed and the foramen magnum opened widely. The arches of the first three cervical vertebrae were also removed. The dura, which at the foramen magnum was found to be three to four millimeters thick, was opened to expose the cerbellum and upper cord. The cerebellar hemispheres had a flattened and atrophic appearance. Near the foramen magnum there was no space for the cisterna magna, as the dura was tightly apposed to the cerebellum and to small tips of flattened cerebellar tonsils that had herniated through the foramen and fitted snuggly against the cord. The uppermost portion of the cord bulged slightly in its posterior aspect, and an aberrant artery curved over it to the right. This part of the cord probably represented an heterotopia, for neither it nor the normal appearing cord below it as far down as the fourth segment felt cystic or yielded fluid on aspiration with a fine needle. The course of the first three pairs of cervical nerve roots was normal but the spinal accessory nerves, particularly the left, were subjected to considerable pressure in the constricted region of the foramen magnum. There were no extensive arachnoid adhesions and it did not appear that the spinal fluid pathways were thus obstructed. The wound was closed but the dura was left open for decompression.

Postoperative Course and Follow-Up.—Recovery from operation was uneventful, and the patient resumed work as a carpenter one month later. Three months after operation he volunteered that he felt steadier on his feet and less clumsy in his upper extremities than he had for several years. No special improvement could be demonstrated on examination.

COMMENT.—The etiology of the platybasia in this case, in view of the coexistence of fused cervical vertebrae, was probably a congenital defect in development of the occipital bone. Rickets has been mentioned by others as a theoretic etiologic factor in platybasia, but its rôle in this case is doubtful.

The majority of the neurologic findings might be attributed to syringomyelia or syringobulbia, and sometimes these congenital conditions have been found to be associated with development defects of the craniovertebral skeleton. However, no syrinx could be discovered at operation and it is likely that at least some of the neurologic changes were the direct result of the cranial deformity.

Case 3.—*Platybasia developing between the fourth and twelfth years in a child with osteogenesis imperfecta. Neurologic signs appeared insidiously after age ten. Operative decompressions of the cerebellum and upper cord was followed by improvement.*

A. M., white, female, age 12, was admitted to the New York Hospital, June 9, 1941, because of cellulitis of the leg.

The girl had first been examined in this hospital for fractured tibia when she was four years old. She had had a spontaneous fracture of the humerus in the first month of life, and had had a succession of at least 20 additional fractures during the succeeding four years. A diagnosis of osteogenesis imperfecta was made and various forms of treatment were employed while she was being followed periodically in the Out-Patient Department. The frequency of fractures diminished and none had occurred after age eight.

At the time of admission to the hospital in 1941, it was brought out in the history that for a year or more speech had had an indistinct and monotonous quality. Also, there was occasional choking and difficulty in swallowing liquids.

Physical Examination.—She was of average size and development for her age. However, the neck was rather short and broad and the head was held with the chin tilted to the left. The head was unusually broad in its biparietal measurement. The sclerae were blue. There was nystagmus on lateral gaze and weak convergence of the eyes. The tongue was protruded to the left, and on the left side was markedly atrophic; there were also fibrillations and atrophy on the right side of the tongue. In addition to thickness, the speech had a nasal quality and the palate moved poorly. Other functions of the head and neck were normal.

There were numerous deformities of the extremities, results of former fractures, but most of these were insignificant. Motility functions were fairly good except for moderate incoordination in point-to-point and rapid rhythmic movements in the upper extremities. Station and gait were not remarkable. The deep reflexes on the right were more active than on the left, and the right plantar response was abnormal. Sensation was everywhere normal but for reduction in position sense in both great toes.

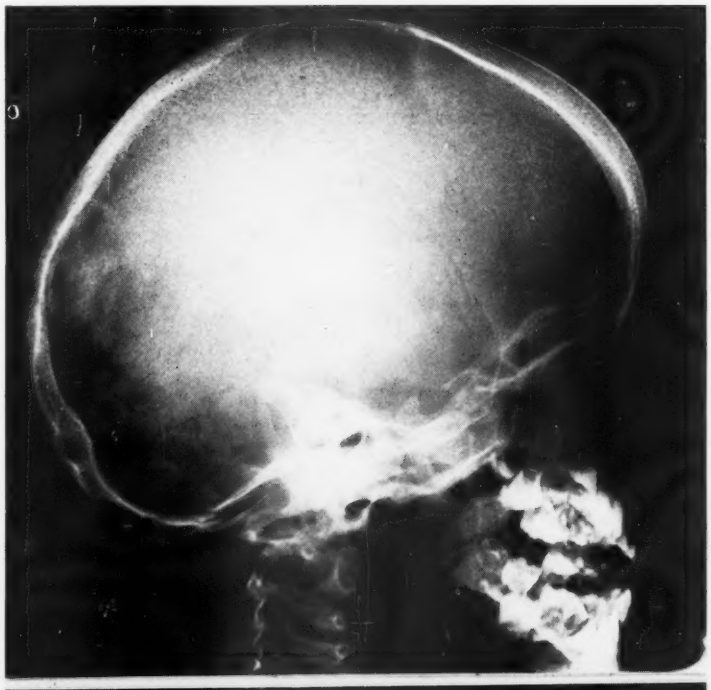
Roentgenograms taken at various times of life had shown the decreased calcium content of all bones—characteristic of osteogenesis imperfecta. The configuration of the skull and cervical spine was normal in 1933 (Fig. 7). The skull plates, taken in 1941 (Figs. 7 and 8), however, showed the vault to be widened and flattened and the floor of the skull behind the sella turcica was invaginated, especially above the atlanto-occipital articulations. The cervical vertebrae were not deformed but the atlas was partly hidden in the invagination of the base of the skull and the odontoid process protruded into the foramen magnum.

Operation.—June 26, 1941: (Fig. 9). Under ether anesthesia, a suboccipital and upper cervical decompression was performed. The skull between the inion and foramen magnum was flat, even slightly concave, and it formed an acute angle with the line of the spinal column, which made the foramen magnum seem unusually deep. The arch of the atlas lay almost completely within the foramen magnum and could be seen clearly only after removal of a segment of the occipital bone and opening of the foramen magnum. Even after removal of the laminae of the atlas and axis the dura at the level of the foramen magnum dipped acutely, as if from long indentation by the arch of the atlas, and certainly appeared to be constricting the structures within. The dura in this region was about two millimeters thick. When the dura had been incised to expose the cerebellum and upper cord, examination showed the cerebellar hemispheres to be generally small and atrophic looking, with very little of the vermis showing and almost complete obliteration of the cisterna magna. The tonsils of the cerebellum were greatly elongated and extended down the spinal cord to the level of the second cervical arch. The left tonsil overlapped the right, suggesting that there was more distortion of the base of the skull on the left side, a supposition which would be compatible with the greater twelfth cranial nerve palsy on the left and right-sided corticospinal tract signs.

There were no adhesions in the region, the cord had a normal appearance, and the course of the cervical nerve roots was normal. The wound was closed, but the dura was left open for decompression.

The bone removed at operation was not remarkable from a gross standpoint, but microscopic examination showed the faulty development characteristic of osteogenesis imperfecta.

FIG. 6



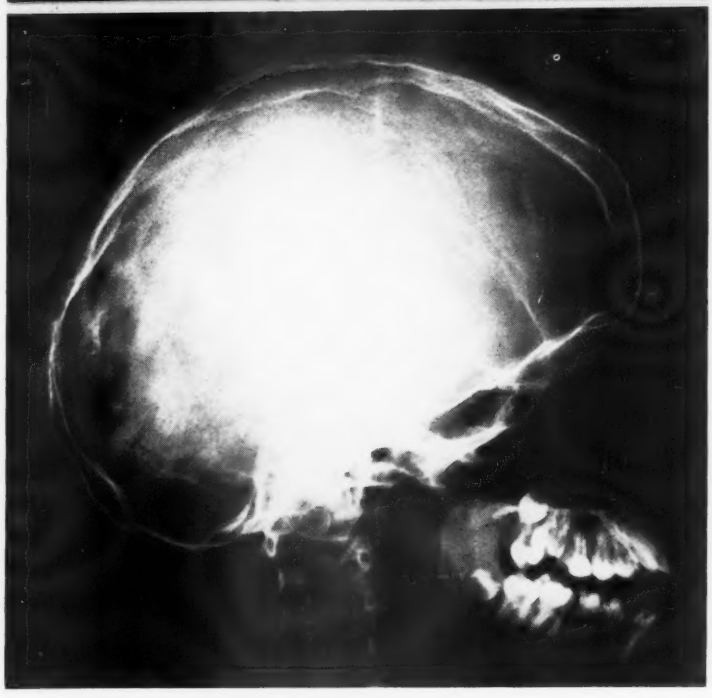


FIG. 7

FIG. 6.—Case 3: Lateral roentgenogram taken at age four (1933) showing poor calcification characteristic of osteogenesis imperfecta but normal configuration in the basilar region.

FIG. 7.—Case 3: Lateral roentgenogram taken at age 12 (1941) showing the acquired platybasia (compare with Figure 7). Note the long, narrow shape of the cranial cavity; the prominence of the petrous ridges; the elevation of the clivus blumenbachii; and the upward displacement of the atlas and axis into the basilar depression.

Postoperative Course and Follow-Up.—Recovery from operation was uneventful. Examination after five months showed a soft, flat area of decompression. There was no appreciable change in speech or in configuration of the tongue. The involvement of the corticospinal tract on the right, and the impaired position sense in the toes could no longer be detected.

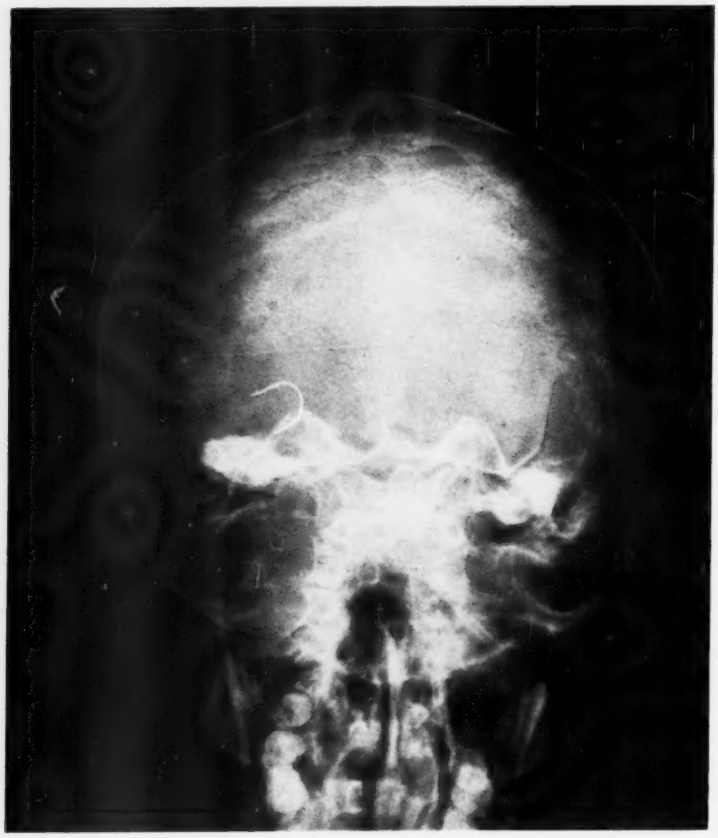


FIG. 8.—Case 3: Anteroposterior roentgenogram taken at age 12 (1941) showing a wide biparietal distance; bulging of the floor of the posterior fossa above the occipito-atlantal articulations; displacement of the odontoid process to the left of the midline and the abnormal appearance of the petrous ridges.

COMMENT.—There is conclusive evidence here that the flattening of the base of the skull was not due to a congenital malformation of the occipital bone. The evidence indicates that the soft, poorly calcified skull had become indented at its base, probably from sheer weight of the growing head upon the spine. The appearance of the skull is, indeed, comparable to what might occur if weight were put upon the top of a flexible skull causing it to elongate, broaden, and indent at its base. The resulting distortion of the brain stem, diminution in the capacity of the posterior fossa, and narrowing of the foramen magnum would amply account for any changes in the nervous system. Because it was assumed that injury to the nervous system would increase,

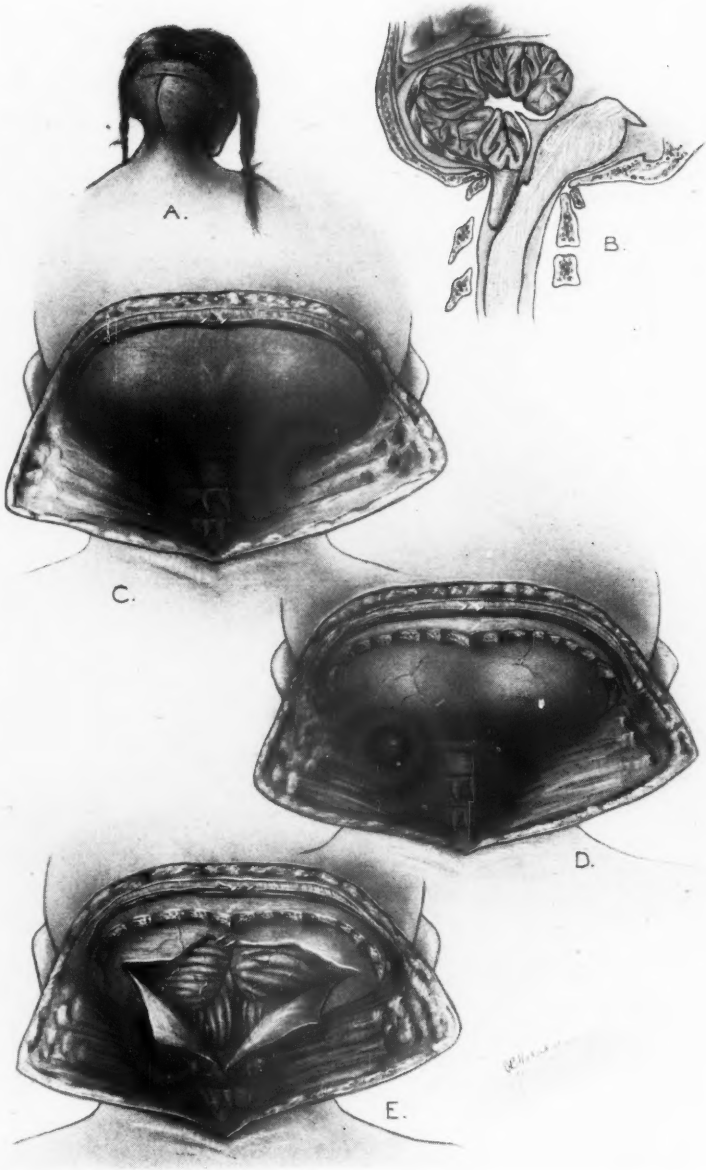


FIG. 9.—Case 3: (A) Type of operative incision. (B) Diagrammatic representation of the nature and effects of the craniovertebral abnormality. (C) View with occipital bone and upper three cervical vertebrae exposed showing concavity of the occipital bone and partial inclusion of the arch of the atlas in the foramen magnum. (D) View after resection of the occipital bone and opening of the foramen magnum showing the arch of the atlas maintaining the constriction of the dura at the foraminal opening. (E) View after resecting the first two neural arches and opening the dura showing the flattened cerebellum; absence of the cisterna magna; and unequal herniation of the cerebellar tonsils into the cervical canal.

decompression was employed even though the existing signs were not advanced. The disappearance of the signs of diseased pyramidal tract on the right side of the body and the return of position sense in the toes are evidences of improvement.

Case 4.—*Platybasia developing in a middle-aged woman with Paget's disease of the skull. Neurologic signs appeared insidiously and advanced steadily over a period of two years. Operative decompression of cerebellum and cord.*

H. C., white, female, age 47, was admitted to the New York Hospital, January 5, 1942, because of headache, vertigo and increasing difficulty in walking. She had been observed periodically in the Out-Patient Department for several months, but entered the hospital when symptoms became severe.

She had been in good health prior to the beginning of enlargement of her head in 1933 (at age 39). A diagnosis of osteitis deformans (Paget's disease) of the skull was made by her physician, and for eight years the head had steadily enlarged. No untoward symptoms appeared until the last two years of this period.

Pain in the back of the head and neck, which was the first symptom, was, in the beginning, mild and unimportant but, later, it became severe. It was induced by coughing, straining, and laughing. It was acute and spread up fan-wise from the back of the neck over the back of the head to the vertex, lasting five to ten minutes.

Gait, which at first was but slightly unsteady, gradually became staggering, and motions in the upper extremities became clumsy. The right arm became weak, and the right foot had dragged during the past year.

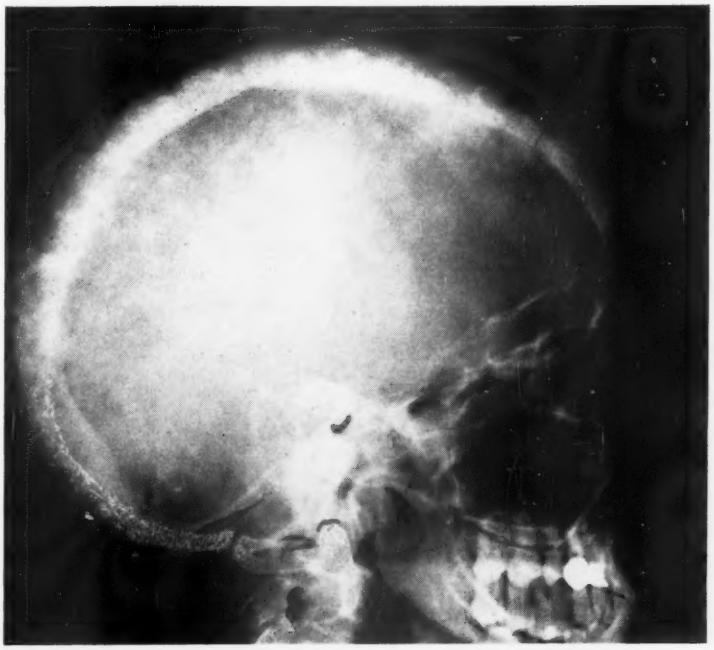
The most recent and distressing symptom was paroxysmal vertigo which was induced by sudden turning of the head. The fear of inducing an attack lead to holding her head in a fixed position.

Physical Examination.—The patient was a slender, fragile looking woman, with a large asymmetrical head and a short wide neck. The neck was held stiffly and the head was moved cautiously. The eyes were slightly prominent and coarse nystagmus developed on lateral gaze which was greater on looking to the right. Hearing was diminished 25 per cent on the left and 15 per cent on the right. The muscles of the tongue showed a moderate degree of atrophy and fibrillations, and when protruded it deviated to the left. Speech was slightly thick and had a nasal quality. The right shoulder drooped. There was clumsiness in rapid rhythmic movements and in point-to-point tests of all extremities. Gait was slow, staggering and broad based. Romberg's sign was positive. There was an hemiparesis of moderate degree involving the right upper and lower extremities. Position sense and two-point discrimination was impaired in the right hand. There was a right hemihypalgesia, not including the face. Position sense was markedly impaired in both feet.

Significant laboratory studies showed normal serum calcium and serum phosphorus but a serum phosphatase of 25 to 30 units (normal is four units). Bleeding and clotting times were normal, but the plasma prothrombin was 18 per cent. The prothrombin value rose to 30 per cent and 70 per cent on successive days on administration of vitamin K (2-methyl-1, 4-naphthaquinone).

Roentgenograms of the skull, taken in 1934 (Fig. 10), showed characteristic changes of osteitis deformans and the early changes of platybasia. Roentgenograms of the skull taken in 1941 (Fig. 11), showed the results of marked indentation of the basi-occipital region and comparison with the earlier films disclosed the progression of this state. There was invagination of the posterior rim of the foramen magnum, narrowing of the posterior cranial fossa, upward and anterior displacement of the atlas and axis. Many other bones in the body showed increase in the trabecular pattern, scattered areas of density and cystic changes but the skull was most prominently involved by the disease and there was no collapse of vertebrae to account for any of the neurologic signs.

FIG. 10.



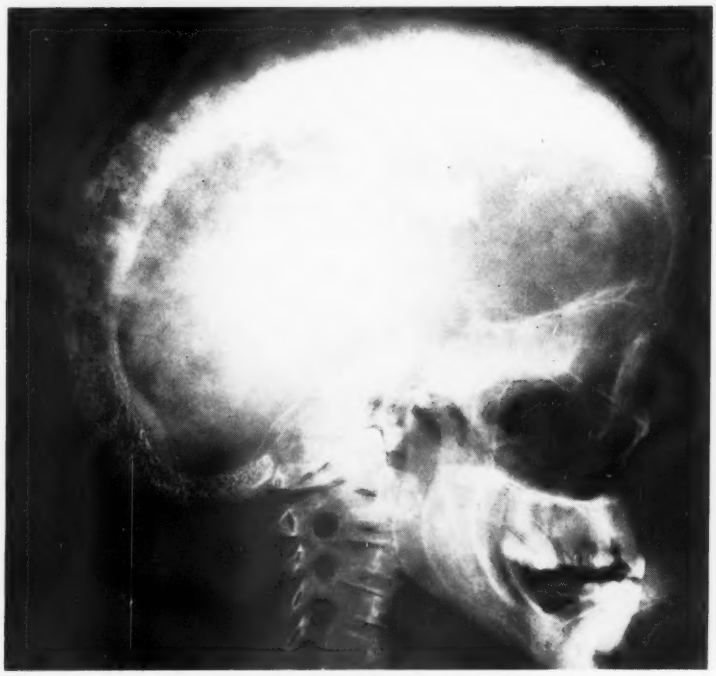


FIG. 11

FIG. 10.—Case 4: Lateral roentgenogram (retouched) taken at age 40 (1934) showing typical osteitis deformans and beginning platybasia which was asymptomatic. (Courtesy of Dr. William Snow.)

FIG. 11.—Case 4: Lateral roentgenogram (retouched) taken at age 47 (1941) showing marked degree of platybasia. By comparison with Figure 11, note the greater narrowing of the posterior fossa and the increased upward displacement of the atlas and axis into the invaginated basiocciput.

Operation.—January 8, 1942: (Fig. 12). Under ether anesthesia, suboccipital and upper cervical decompression was performed. The operation was attended by an unusual amount of bleeding from the soft tissues and the bone, which required assiduous atten-

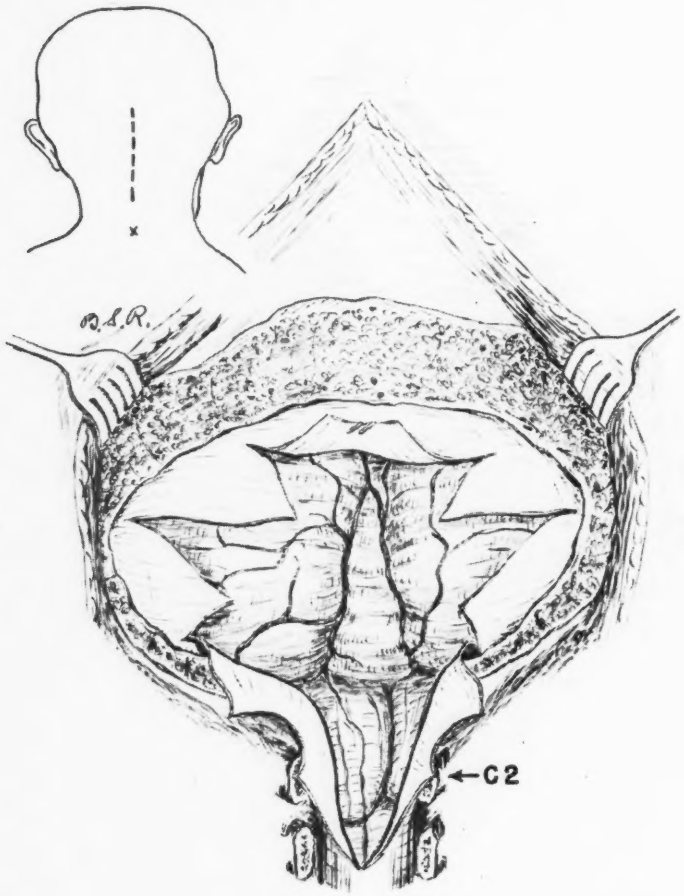


FIG. 12.— Case 4: Operative sketch showing the cerebellum and upper cervical canal exposed. Note the flattening of the cerebellar hemispheres; the wide vermis grooved by the narrow foramen magnum; the absence of the cisterna magna; and the marked and unequal herniation of the cerebellar tonsils into the cervical canal. The inset shows the type of operative incision.

tion to hemostasis. The skull was from two to four centimeters thick, and so soft that the tip of a forceps could be plunged through it. The bone of the vertebral spines and arches was only slightly softer than normal. The arch of the atlas lay almost completely within the foramen magnum and appeared to be indenting the posterior rim of the foramen. The neural arches of the second and third cervical vertebrae were removed.

A fair-sized window in the occipital bone was rongeured and curetted away. After the foramen magnum had been widely opened it was then possible to remove the arch of the atlas. In this region the dura was slightly thickened and gave the appearance of constricting the structures lying within it.

When the dura had been incised to expose the cerebellum and cord it was seen that the cerebellar hemispheres were flattened, the vermis was unusually wide and the cerebellar tonsils were herniated into the spinal canal to the upper level of the third cervical vertebra. The left tonsil was longer, more prominent and overlapped the right, suggesting greater distortion of the base of the skull on the left, a supposition which would be compatible with the right hemiparesis. There was no cisterna magna but after the dura had been opened an exchange of subarachnoid fluid between the cranial and spinal cavities could be demonstrated. There were no adhesions in the region, and what could be seen of the cord appeared normal. The wound was closed but the dura was left open for decompression.

Microspocic examination of the bone removed at operation showed the changes typical of osteitis deformans.

Postoperative Course.—Recovery from operation was complicated only by edema of the neck during the first three days. Early indications pointed to improvement in all symptoms but the postoperative period is too short to evaluate results.

COMMENT.—As in Case 3, this is unquestionably an example of acquired platybasia. The extreme softness of the skull and the increase in the weight of the head are conditions well suited to the development of platybasia. It is likely that this basilar deformity occurs in some degree in the majority of people with advanced osteitis deformans of the skull. The extreme narrowing of the posterior cranial fossa and of the foramen magnum, which can be seen on roentgenogram, and which was, in this case, demonstrated at operation, possesses many possibilities for the production of neurologic symptoms. Whereas in the past the headache and various cranial nerve palsies frequently found to be associated with osteitis deformans have been largely interpreted as symptoms to be expected from such great thickening of the skull, it may develop that often such symptoms are the direct or indirect result of platybasia.

Decompression operation in patients with osteitis deformans is perhaps more hazardous than in others because these patients, in general, do not stand operations well, and there is the added difficulty in dealing with the increased vascularity of the bone and adjacent soft tissues. But this should be no contraindication to the operation if the signs and symptoms present warrant it.

DISCUSSION

Comparatively few references to platybasia have appeared in the Anglo-American literature[1, 2, 3] and the deformity, though mentioned in European literature as far back as 1844,[4] has never received the wide attention it deserves. Boogard,[5] in 1865, Virchow,[6] in 1876, and Grawitz,[7] in 1880, described in detail the changes in the skull. Homén,[8] in 1901, correlated neurologic observations with postmortem study of the effects of pressure of the craniovertebral deformity upon nervous structures. Schüller,[9] in 1911, by roentgenologic examination of the skull, first demonstrated basilar impression in

the living and, with an appreciation of the effects of the deformity upon the nervous system, described clinical examples having manifestations of medullary compression, cerebellar compression and cranial nerve involvement. Additional clinicopathologic studies have been reported by Sinz,[10] Kecht,[11] Krause,[12] Merio and Risak,[13] Juhlin-Dannfelt,[14] Ebenius,[15] and in this country by Chamberlain,[1] Gustafson and Oldberg,[2] and List.[3]

The etiology of the craniovertebral deformity has from the time of its early description been ascribed largely to congenital maldevelopment or hypoplasia. List has described in detail a variety of anomalies of the occipital bone, atlas and axis in terms of the embryonic development of these structures. Case 1, in which there was synostosis of the atlas and occiput, is an example of congenital maldevelopment. Possibly Case 2 is also an example of prenatal maldevelopment, particularly in view of the synostosis of two of the cervical vertebrae but without this, one would give more consideration to the possible rôle of rickets in the patient's early life.

Most authors have suspected, with meager proof, that platybasia may be acquired as a result of such diseases as osteomalacia, rickets, syphilis, osteitis deformans and chronic increased intracranial pressure. Cases 3 and 4 are the only examples, that have come to my attention, in which the development of platybasia with accompanying neurologic symptoms has been demonstrated in the course of such disease. In each of these cases (one of osteogenesis imperfecta, and the other of osteitis deformans) the comparison of roentgenograms of the skull, taken years apart, shows this development. In one instance the patient was a growing child, in the other a mature woman. A condition comparable in its effects may occur following fracture or dislocation at the occipito-atlanto-axial region; such a case has been reported from this clinic.[16]

Much depends upon roentgenologic examination in the diagnosis; for although there are many characteristic clinical features of the condition the final diagnosis in any case rests on the demonstration of the craniovertebral deformity by roentgenograms. Schüller[17] has ably enumerated the characteristic changes that may be seen roentgenographically, and most of the recent contributors have added to this aspect of the subject.[15, 1, 2, 3] The roentgenograms reproduced herewith emphasize most of the essential abnormalties.

The various roentgenographic changes seen in platybasia may be summarized as follows. In the lateral view may be seen: Rudimentary development of the occipito-atlanto-axial region; dislocation of the atlas and axis upward and forward; projection of the odontoid process above a line drawn from the posterior rim of the foramen magnum to the hard palate; upward displacement of the clivus blumenbachii; invagination of the posterior rim of the foramen magnum; high prominent position and more or less circular shadow of the petrous portions of the temporal bones; and, a relatively long, vertically narrow appearance of the cranial cavity.

In the sagittal views taken in the anteroposterior direction and in the "occipital projection" may be seen: Asymmetry of the posterior cranial fossa and of the foramen magnum; upward dislocation of the atlas and axis; abnormal relation of the positions of the atlas and odontoid process to the lumen of the foramen magnum; abnormality of the atlanto-occipital articulations; prominence, unusual shape and asymmetry of the petrous portions of the temporal bones; and broad biparietal measurement of the skull.

All patients with platybasia possess more or less the same appearance above the shoulders (Fig. 1). The neck is distinctly short and thick, the hair line at the back is low. The position of the head in profile is somewhat forward of the perpendicular axis of the body and in front view is usually tilted or turned. This appearance is due largely to the craniovertebral deformity and need not be accompanied by limitation of movements of the head, pain or neurologic changes.

Only when neurologic symptoms occur, does platybasia become anything more than a developmental curiosity. It has been emphasized by other contributors to this subject that in congenital platybasia it is but reasonable to expect the presence of other congenital abnormalties. [3, 18] Thus, the occurrence of symptoms of syringomyelia in conjunction with platybasia may be only the result of a congenital "dysraphic" state which implicates both the nervous and skeletal systems; developmental disturbances in numerous midline structures have been found to accompany syringomyelia.[19] To accept this as the only explanation for the coexistence of platybasia and syringomyelic symptoms is failure to recognize the direct etiologic relationship that may exist between the skeletal deformity and numerous associated changes in the nervous system. In the case of sensory dissociation of syringomyelic nature, for example, the changes may be the result of bony compression of the ventrolateral tracts of the medulla. As a result of experience with a patient—in whom no syringomyelia was found at operation, and improvement followed operative decompression, Chamberlain stressed particularly "the tendency for basilar impression to masquerade as syringomyelia"; Case 2 is comparable to this. On the other hand, it is justly argued by Gustafson and Oldberg that when, in the presence of platybasia, there exists an hydromyelic state of the central canal, the two conditions probably exist as cause and effect.

Thus, in platybasia acquired changes in the nervous system may occur singly or in combination as a result of (1) direct pressure upon the brain stem, cranial nerves, cerebellum and cervical cord, (2) embarrassment of the blood supply of these structures by adhesions or compression, or (3) interference with cerebrospinal fluid pathways.

The flattened appearance of the cerebellar hemispheres, the absence of the cisterna magna, the protrusion of the cerebellar tonsils into the spinal canal, plus the dural constriction at the region of the foramen magnum demonstrated at operation, indicate the degree of general compression exerted upon these

nervous structures by the narrowed confines of the posterior fossa. Hydrocephalus of varying degree might be expected to accompany this state of affairs, and has been demonstrated by some observers.[2, 14]

The deformity of the nervous system found to accompany platybasia has been likened to the Arnold-Chiari deformity.[2, 3] The latter is not a well defined malformation but is generally regarded as a downward elongation of the cerebellum and brain stem into the cervical portion of the spinal canal associated with spina bifida and myelomeningocele; hydrocephalus is frequently present as a result of obstruction of the fourth ventricle.[20, 21] While the appearance of the brain and cord may in some respects be similar in the two conditions the mechanism producing the one is the antithesis of that producing the other. In platybasia the structures may be thought of as being pushed through the foramen magnum, whereas in the Arnold-Chiari malformation, they are pulled. The simple proof of the difference exists in the relatively normal course of the cervical nerve roots in the former and the abnormal upward course of the roots to their foramina in the latter.

This brief allusion to some aspects of the effects of platybasia serves to emphasize that any number of neurologic symptoms may result, depending upon the particular parts of the central nervous system that are impinged upon or otherwise disturbed by the craniovertebral deformity. When the diagnosis has been established, operative decompression should be given thoughtful consideration. In the past, it has been assumed that platybasia and its associated neurologic symptoms were solely a medical curiosity, to be recognized in order that needless operation could be avoided.[14, 17] Obviously, no operative procedure can rectify the invagination of the base of the skull or the displacement of the atlas and axis, but the removal of part of the squama of the occipital bone, the opening of the foramen magnum and the removal of one or two neural arches serves to enlarge the narrowed space occupied by the brain stem, cerebellum and upper cervical cord. Neither can it be expected that operative decompression will result in alleviation of all neurologic changes but the evidence indicates that improvement of existing symptoms and prevention of progressive changes may be hoped for.

Besides the four cases reported herewith, 12 others, having deliberate decompressive operations, have been reported.[1, 2, 3] From an analysis of these cases two principles stand out: (1) That the dura must be opened to obtain satisfactory decompression; and (2) that the less intradural manipulation of nervous structures the better. Personal experience has led to the belief that simple operative decompression is not hazardous and has much to recommend it when contrasted with the unfavorable prognosis of advanced compression of the brain and cord.

CONCLUSIONS

Platybasia is a craniovertebral anomaly, congenital or acquired, that may be accompanied by changes in the central nervous system that result either directly from pressure or indirectly from the effects of the bony deformity.

Platybasia causes a characteristic appearance of the head and neck, and the neurologic symptoms masquerade as neoplasm of the posterior fossa or upper cervical canal, hydrocephalus, syringomyelia, syringobulbia, multiple sclerosis or spinocerebellar disease. The final diagnosis rests upon the roentgenographic demonstration of invagination of the basioccipital region of the skull and the upward displacement of the upper cervical vertebrae into this depression, producing narrowing of the posterior cranial fossa, foramen magnum and upper cervical canal. When neurologic symptoms are disabling or progressive, operative decompression of the cerebellum and upper cord is beneficial in improving or staying the progress of the nervous changes. Simple operative decompression is not unduly hazardous.

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DISCUSSION: DR. IRA COHEN (New York) said that the cases presented by Doctor Ray represented almost a medical curiosity, inasmuch as platybasia is very rare, though not so rare as would be assumed from the paucity of cases reported in the literature. Cases are missed, first, because roentgenograms, if taken, are not carefully examined; and, secondly, for the more important reason, that in cases diagnosed as syringomyelia, intrinsic disease of the cord, or multiple sclerosis, roentgenograms of the upper cervical spine and skull are not taken and studied. The term "platybasia" was brought to the forefront after an article by Chamberlain a few years ago. Very probably "basilar invagination" is a more descriptive term. The platybasia itself is partially responsible for symptoms, but the majority of the symptoms are due to the deformity about the foramen magnum in which the skull and vertebrae combine. The symptoms are caused by compression, that is, narrowing of the foramen magnum, the pressure of the odontoid process, and very often there is thickening of the dura and bands of dura in this region, plus herniation of the cerebellum into the spinal canal.

Doctor Cohen felt that Doctor Ray's third case was of particular interest because of the fact that the bony deformity was seen to develop while the patient was under observation, also, the onset of neurologic signs was seen to occur. There is no really adequate explanation for the development of symptoms, in the second, third, fourth, or even fifth decade, from a condition which is congenital. If the bony deformity is static then, perhaps, one must assume, with Chamberlain, that the young nervous system is more able to withstand the resulting pressure than is that of the adult. Otherwise, there is no real explanation of why symptoms develop late in life in some individuals. In this young girl, actual positive proof is offered that, under observation, there can be a change in the contour about the foramen with the onset of symptoms. Rickets, osteomalacia and many other conditions have been mentioned as a consequence. Doctor Cohen said he knew of no other case where such proof had been demonstrated.

The very appearance of the patients as they walked into the room was almost diagnostic.

EXTRALARYNGEAL DIVISION OF THE RECURRENT LARYNGEAL NERVE*

ITS SIGNIFICANCE IN VOCAL CORD PARALYSIS

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VOCAL CORD PARALYSIS, if it is unilateral, is a serious complication of thyroid surgery and, if bilateral, constitutes a surgical calamity. The patient is generally told after a unilateral cord injury that the voice will return and no treatment is necessary. In the bilateral cord injuries, if the cords are in adduction, a tracheotomy may have to be performed as a life-saving measure, and efforts made at a later date to restore the function of the cords. This may be attempted by some plastic procedure on the cords or else by exposing the recurrent laryngeal nerve with an attempt at anastomosing the severed ends, as advocated by Lahey and Hoover,[10] or anastomosing the distal end of the recurrent laryngeal to the descending branch of the hypoglossal nerve, as advocated by Frazier.[5]

During the past three years, in the Thyroid Clinic at Post-Graduate Hospital, our interest has been aroused by a conservative method of treatment in vocal cord paralysis that was instituted by Dr. Charles O. Fiertz, of the Neurologic Department. Lahey and Hoover,[10] in 1938, advised exploring the neck of patients with vocal cord paralysis within the first three months of the operative injury. They felt that if the patient had gone longer than three months there was a greatly diminished chance of nerve regeneration and also a chance of fixation of the arytenoid muscles due to atrophy and fibrosis. Jackson,[7] in 1937, states that a vocal cord paralysis of six months' duration is permanent and can never be restored. With such opinions one would look with skepticism upon any method of conservative treatment being employed to restore a vocal cord that has been functionless for many months. While discussing, in our clinic, the advisability of exploring the neck of a patient with a vocal cord injury which had gone approximately one year, the conservative treatment, as referred to in this presentation, was advised by Doctor Fiertz. The optimum time for resuturing the nerve had long since past, according to the teaching of Lahey and Hoover, and the cord was permanently paralysed, by Jackson's concept of vocal cord paralysis. In view of their opinions we felt that conservative treatment would do no harm, and one of the patients whom we present this evening illustrated our first experience with galvanic and faradic stimulation, as carried out by Doctor Fiertz with the aid of Dr. A. F. Laszlo, of the Otolaryngology Department. A detailed discussion of the exact technic is being presented before the New York Neurological Society by Doctor Fiertz. It will suffice in this presentation to say the nerve is stimulated by an uninterrupted cathode application and the

* Read before the New York Surgical Society, April 8, 1942.

muscles of the vocal cords by faradic and galvanic stimuli. If the vocal cord does not respond to the faradic and galvanic stimuli on the first examination the prognosis for ultimate improvement is poor, but not necessarily hopeless. Even if no response is elicited on the first examination, treatment is rendered for six weeks and if after that time no response is obtained the nerve is considered permanently injured. A very small percentage of this group will respond to treatment. The favorable cases are those showing movement in the vocal cords to faradic and galvanic stimulation on the first visit, which indicates the motor activity is not completely lost.

We have had occasion to refer 19 patients for electrical treatment during the years 1940 and 1941. It should be stated that all these nerve injuries were by no means incurred at Post-Graduate Hospital, but we can also add that the percentage of injuries occurring after operations performed by the General Surgical Staff far exceeded 3 per cent. This figure, given by Lahey and Hoover,[10] was the average, and was high compared to their clinic, 1.6 per cent before demonstrating the recurrent laryngeal nerve, and 0.3 per cent after demonstrating the recurrent laryngeal nerve at operation.

After we had seen definite clinical improvement in vocal cord paralysis following the electrical stimulation, the problem was discussed by the authors, and we felt the explanation of the clinical improvement was due to incomplete loss of the motor activity of the recurrent laryngeal nerve, that many nerves had two branches, one branch would be injured while the other remained intact. The intact branch was so reduced as to represent one-third or one-half the original caliber of the nerve and, therefore, only able to function when assisted by artificial stimulation to regain motor control over the injured muscles. Therefore, we wish to report our findings on a group of dissections, made in the Anatomical Laboratory at Post-Graduate Hospital, which were undertaken by the authors, and consisted of ten cadavers, and the exposure of 19 nerves (Tables I and II). We wish to report the ob-

TABLE I

POST-GRADUATE HOSPITAL SERIES

Weeks and Hinton

Ten Cadavers—19 Nerves

78 Per Cent Divided Extralaryngeally

RIGHT SIDE

Division of Nerve	Extralaryngeal Division, in Cm.	Division in Relation to Thyroid Gland	Relation of Nerve to Inferior Thyroid Artery	Relative Diameter of Divisions
Two.....	4 cm.	Lower pole	Posterior	65%—35%
Two.....	2 cm.	Middle third	Posterior	75%—25%
Single*.....	.....	.....	Anterior	.....
Two.....	2.5 cm.	Lower pole	Anterior	65%—35%
Two.....	1 cm.	Upper third	Anterior	50%—50%
Two.....	2.5 cm.	Middle third	Anterior	65%—35%
Three.....	2 cm.	Middle third	Anterior	50%—25%—25%
Two.....	2.5 cm.	Lower third	Posterior	75%—25%
Two.....	3 cm.	Lower pole	Anterior	50%—50%

* Single: Between branches of inferior thyroid artery.

TABLE II

POST-GRADUATE HOSPITAL SERIES

Weeks and Hinton

Ten Cadavers—19 Nerves

78 Per Cent Divided Extralaryngeally

LEFT SIDE

Division of Nerve	Extralaryngeal Division, in Cm.	Division in Relation to Thyroid Gland	Relation of Nerve to Inferior Thyroid Artery	Relative Diameter of Divisions
Two	4 cm.	Lower pole	Posterior	65%—35%
Two	5 cm.	Lower pole	Posterior	65%—35%
Single*	.....	.....	Anterior	.....
Destroyed	.....	.....	.....	.....
Two	1 cm.	Upper third	Anterior	50%—50%
Two	2.5 cm.	Middle third	Anterior	50%—50%
Two	2.5 cm.	Middle third	Posterior	50%—50%
Two	3 cm.	Lower pole	Posterior	50%—50%
Two	3 cm.	Middle third	Anterior	50%—50%

* Single: Between branches of inferior thyroid artery.

servations made in the Anatomical Laboratory of New York University by Mr. John Stewart and Mr. Roger Moore, who are fourth year medical students. They did this as a favor to the authors, checking our observations on 18 cadavers, with 24 nerve exposures (Tables III and IV). In the dis-

TABLE III

NEW YORK UNIVERSITY SERIES

Stewart and Moore

Eighteen Cadavers—24 Nerves

78 Per Cent Divided Extralaryngeally

RIGHT SIDE

Division of Nerve	Extralaryngeal Division, in Cm.	Division in Relation to Thyroid Gland	Relation of Nerve to Inferior Thyroid Artery	Relative Diameter of Divisions
Two	2.2 cm.	Lower third	Anterior	65%—35%
One	.....	.....	Anterior	.....
Two	1.5 cm.	Middle third	Anterior	50%—50%
Single	.....	.....	Anterior	.....
Destroyed	.....	.....	.....	.....
Two	2.0 cm.	Junction of middle and lower third	Posterior	50%—50%
Destroyed	.....	.....	.....	.....
Destroyed	.....	.....	.....	.....
Single	.....	.....	Posterior	.....
Destroyed	.....	.....	.....	.....
Two	2 cm.	Junction of middle and lower third	.....	75%—25%
Destroyed	.....	.....	.....	.....
Two	2.5 cm.	Lower third	Anterior	65%—35%
Two	2.0—2.0 cm.	Middle third	Posterior	65%—35%
Two	3.5—2.5 cm.	Lower pole	Posterior	65%—35%
Destroyed	.....	.....	.....	.....
Destroyed	.....	.....	.....	.....
Destroyed	.....	.....	.....	.....

sections at Post-Graduate Hospital we found that 78 per cent of the recurrent laryngeal nerves divided extralaryngeally while at New York University it was the same. Many of these nerves started their division at the level of the lower pole of the gland. These findings would, obviously, explain how

it is possible to injure one extralaryngeal branch at operation, with complete paralysis of the vocal cord on the corresponding side, and making it possible for electrical stimulation to restore the cord to a state of functioning after a

TABLE IV

NEW YORK UNIVERSITY SERIES

Stewart and Moore

Eighteen Cadavers—24 Nerves

78 Per Cent Divided Extralaryngeally

LEFT SIDE

Division of Nerve	Extralaryngeal Division, in Cm.	Division in Relation to Thyroid Gland	Relation of Nerve to Inferior Thyroid Artery	Relative Diameter of Divisions
Two.....	1.6 cm.	Lower third	Anterior	65%—35%
Two.....	3.0 cm.	Inferior pole	?	50%—50%
		Junction of middle and lower third	Posterior	50%—50%
Single.....	.....	.....	Anterior	.....
Two.....	1.0 cm.	Middle upper half	?	65%—35%
Destroyed.....	.....	.....	.....	.....
Two.....	1.25 cm.	Middle third	Posterior	65%—35%
Two.....	2 cm.	Inferior pole	Artery splits the 2 branches	50%—50%
Destroyed.....	.....	.....	.....	.....
Two.....	2 cm.	Junction of middle and lower third	Posterior	50%—50%
Two.....	2.5 cm.	Inferior pole	Artery splits branches of nerve	75%—25%
Two.....	1.5 cm.	Middle third	Posterior	65%—35%
Single.....	.....	.....	Anterior	.....
Destroyed.....	.....	.....	.....	.....
Two.....	2.25 cm.	Inferior pole	Posterior	50%—50%
Three.....	2.5 cm.	Inferior pole	Posterior	40%—20%
Single.....	.....	.....	Posterior	.....
Two.....	2 cm.	Middle and lower third	Posterior	50%—50%

definite period of paralysis; meaning one that has existed in terms of years rather than in terms of months. Figure 1 is an actual photograph from a dissection showing the nerve dividing with one branch 65 per cent, and the other 35 per cent of the original caliber of the nerve.

When one reviews the anatomy of the recurrent laryngeal nerve, and recent articles which include numbers of dissections of the laryngeal nerve, one wonders why this point has not been stressed before. In all probability the nerves were not completely dissected as they entered the larynx at the inferior horn of the thyroid cartilage. The authors had other points of interest in their dissections; for instance, one point of controversy has been the position the recurrent laryngeal nerve had in relation to the inferior thyroid artery. Although several articles have been published to clarify this point, after anatomic dissections, there is considerable difference of opinion. Fowler and Hanson,[6] in a very excellent article on 200 dissections, state that in 65.5 per cent the nerve was posterior to the artery, and in 26 per cent anterior, and in 8.5 per cent it lay between the branches. Berlin and Lahey,[2] in 22 dissections, state the nerve is anterior to the artery in 18 cases on the right side, and posterior to the artery in 19 cases on the left side. Ziegelman,[13]

in 42 dissections, reports the nerve anterior to the artery in 17, and posterior to the artery in 12, and between in 13; while our own dissections essentially confirm those of Ziegelman. There have been three references made in the literature to the nerve having extralaryngeal branches. Berlin[1] reports two instances, in 70 total thyroidectomies, where the recurrent laryngeal nerve branches at the inferior pole of the gland, and in one instance the anterior branch was divided, with paralysis of the vocal cord on the same side. Nord-

FIG. 1.—Photograph showing division of recurrent laryngeal nerve at the lower third of gland. Branches 65-35 per cent of the original caliber.

land,[11] in 31 cadavers, found in two instances branches between the inferior thyroid artery on the left side, and in two instances branches between the inferior thyroid artery on both sides. Lahey,[9] in an article advocating the dissection of the recurrent laryngeal nerve in subtotal thyroidectomies, has an illustration in which the recurrent laryngeal nerve branches extralaryngeally, and says that he has repeatedly seen the extralaryngeal division of the nerve in the course of dissecting the nerve during thyroidectomies, which at that time had numbered over 3,000 cases.

How easily the recurrent laryngeal nerve is injured by handling or manipulation of the gland, or by traction, is important. If it is easily injured then one would have a logical explanation of why a nerve would refunction after months, or perhaps years, with the aid of electric stimulation. Crile,[4] as recently as 12 years ago, stated that the slightest direct or indirect pressure on the recurrent laryngeal nerve interfered with nerve conduction and immediately changed the voice. Doctor Fiertz feels as Crile did, and doubts our anatomic explanation of the clinical improvement. The experimental work of Judd, New and Mann[8] contradicts this in their work on dogs; they put traction on the nerve, several times greater than one would use in performing a thyroidectomy, without producing any voice changes. Lahey,[9] more recently, has confirmed the observations of Judd, *et al.*, and states, in

over 3,000 dissections, he does not believe the nerve was injured by handling during such a procedure.

It is important to stress the motor function of the superior laryngeal nerve as causing voice change in thyroid operations. It is a well-known fact that the external branches of the superior laryngeal nerve, which accompanies the superior artery, supply the cricothyroid muscle. The cricothyroid muscle is a tensor muscle of the larynx, and its paralysis results in a hoarseness or a voice that becomes tired after moderate talking. The internal branch of the superior laryngeal nerve also has a definite motor function, and supplies the arytenoids which are adductors in function. Nordland,[11] in 19 laryngeal dissections, found the arytenoid muscles were supplied by the internal branch of the superior laryngeal nerve in 18 specimens, and in one specimen a branch of the recurrent laryngeal nerve and the internal branch of the superior laryngeal supply the arytenoid muscles.

The percentage of vocal cord paralysis in unoperated goiters is one of extreme interest, particularly when the postoperative incidence is given in the literature as only from 0.3 to 3 per cent by different authors. The preoperative paralysis, which one assumes is due to pressure on the nerve, is given as 5 to 10 per cent by Wolfer, who is quoted by Reinhoff.[12] If so, preoperative examination of the vocal cords in all goiter patients is essential. Obviously, any patient who has had a thyroid operation of any kind should have the vocal cords examined, even though the operation has been inferior or superior pole ligation. In our clinic we have not encountered any preoperative vocal cord paralysis in unoperated cases. Lahey[8] has stated that the routine laryngeal examination in unoperated cases is such a burden, and so seldom of any clinical significance, that he has stopped doing it routinely. That does not mean that in case of a large goiter that produces pressure symptoms, the patient should not have preoperative vocal cord examination, but in the hyperplastic goiter and small nodular goiter it seems of very little value. One should remember that Wolfer's statement for preoperative injury to the vocal cords was made in 1879, and he was observing large goiters, and many of them were substernal.

The point at which the nerve is usually injured is generally agreed to be at its entrance into the larynx in the region of the inferior horn of the thyroid cartilage. This site has been stressed by Blalock and Crowe,[3] and our clinical observations confirm their statement. That being the case, one can readily see how difficult it is to prevent injury to one of the branches of the recurrent laryngeal nerve by exposing the nerve at the inferior thyroid artery, unless the nerve is completely dissected free and the dissection carried to the larynx, thus making sure that both branches are visualized. This is, anatomically, rather difficult with the thyroid gland in place. Therefore, one might question the wisdom of dissecting the recurrent laryngeal nerve as a routine operative procedure. The extralaryngeal division of the recurrent laryngeal nerve, in a high percentage of anatomic dissections, can explain

the favorable results in many of our cases and can also explain why, in a definite percentage of cases, failure to improve will be encountered due to the fact that the nerve has no extralaryngeal branches.

COMMENT

The conservative treatment for vocal cord paralysis, that has been discussed in this presentation, seems to have a definite anatomic basis, and a certain degree of success should be anticipated from such a procedure. In 19 cases there were two bilateral paralyses from roentgenotherapy. One of these patients had worn a tracheotomy tube for seven years before treatment was instituted. Within three months after instituting treatment the tracheotomy tube was removed, and the patient has a normal voice, as was seen from the case presented this evening. The other patient had gone three years before treatment was started and she has a normal voice at the present time. These cases fall in a different category from operative injuries.

The mechanism is an ankylosis of the cricoarytenoid joint, due to the severe inflammatory reaction from the roentgenotherapy. There were six bilateral and 11 unilateral operative cases. Of the six bilateral, two were complete failures, three were greatly improved and one was cured. Of the 11 unilateral injuries, two were cured, four were greatly improved, and five were definitely benefited.

From our case presentations it would seem there is a definite place for conservative treatment of vocal cord paralysis, as rendered by Doctor Fiertz. In those cases with an extralaryngeal division of the recurrent laryngeal nerve, if the impulses have not been completely interrupted, a cure, in the sense of a normal functioning vocal cord, can be anticipated in a certain percentage of operative injuries.

A vocal cord paralysis that has existed beyond the six months' period does not represent a permanent paralysis, as stated by Jackson, according to our observations in the above 19 cases.

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REPAIR OF DIRECT INGUINAL HERNIA WITH OSTEOPERIOSTEAL TRANSPLANT

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One out of every five direct inguinal herniae operated upon has recurred. This high percentage of surgical failures has not gone unchallenged. Many original procedures and modifications of the old ones have been tried, but none of these have materially reduced the number of recurrences. The excellence attained in the surgery of indirect inguinal herniae can be traced to the successful anatomic reconstruction of the defect in the abdominal wall. The failure to achieve comparable success in the direct type must be attributed to the incomplete reconstruction of the defect in the abdominal wall. In 1938, Veal and Baker[1] described an operation for direct hernia based upon the construction of a new floor of Hesselbach's triangle with an osteoperiosteal transplant. The results obtained with this new procedure in 85 cases have been so encouraging that I feel a follow-up report is now warranted.

In order to make clear the principle of the osteoperiosteal repair it is necessary to review the normal anatomy of the structures involved, and show how these structures are altered when a direct hernia develops. A direct hernia pierces the abdominal wall through Hesselbach's triangle. This triangle is bounded laterally by the deep inferior epigastric vessels, medially by the edge of the rectus muscle, and inferiorly by the inguinal ligament. These boundaries are of little importance except for the point of diagnosis. The structures forming the floor of this triangle are most important because a hernia cannot develop if they are normally formed and remain intact. The floor of Hesselbach's triangle is made up of several layers. From within outward are the peritoneum, extraperitoneal fat, transversalis fascia, and the conjoined tendon. The peritoneum should not be considered as a buffer. The transversalis fascia is a thin aponeurotic membrane which lies between the inner surface of the transversus abdominis muscle and the extraperitoneal fat. In the inguinal region it become closely adherent to the aponeurosis of the transversus. The conjoined tendon is a thick, inelastic fibrous sheet which forms a solid barrier in the floor of the Hesselbach's triangle. This tendon is formed by the union of the tendons of the obliquus internus and transversus abdominis muscles. The tendinous portions of these muscles become fused and continue as a single broad sheet medially toward the rectus sheath and downward toward the pubis. It is inserted into the rectus sheath, the crest of the pubis, and the ascending ramus of the pubis. Its attachment to the ascending ramus is along the pectineal line and extends laterally to the deep inferior epigastric vessels (Fig. 1). The conjoined tendon is also loosely joined to the inguinal ligament, but this is not a true anatomic insertion.

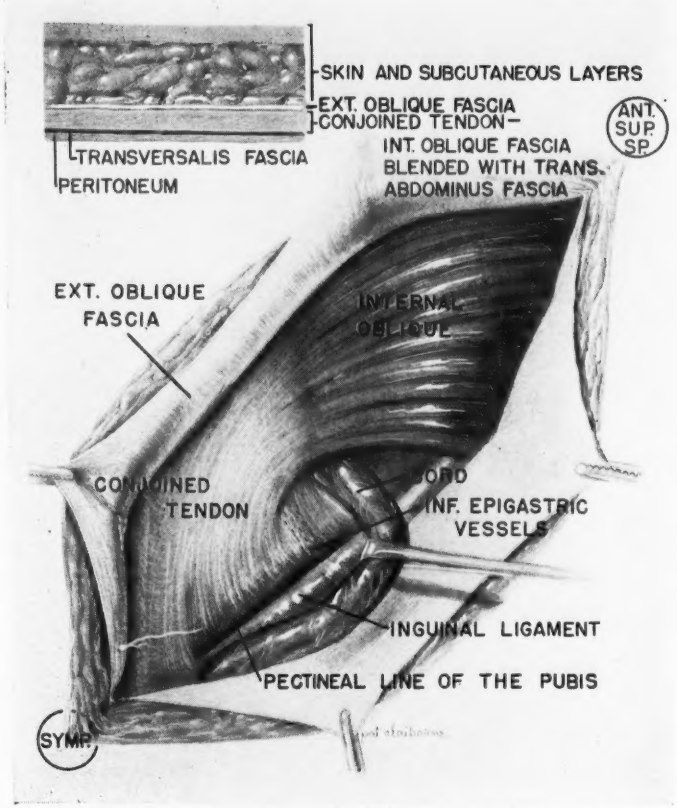

FIG. 1.—Drawing showing the normal relations and attachments of the conjoined tendon.

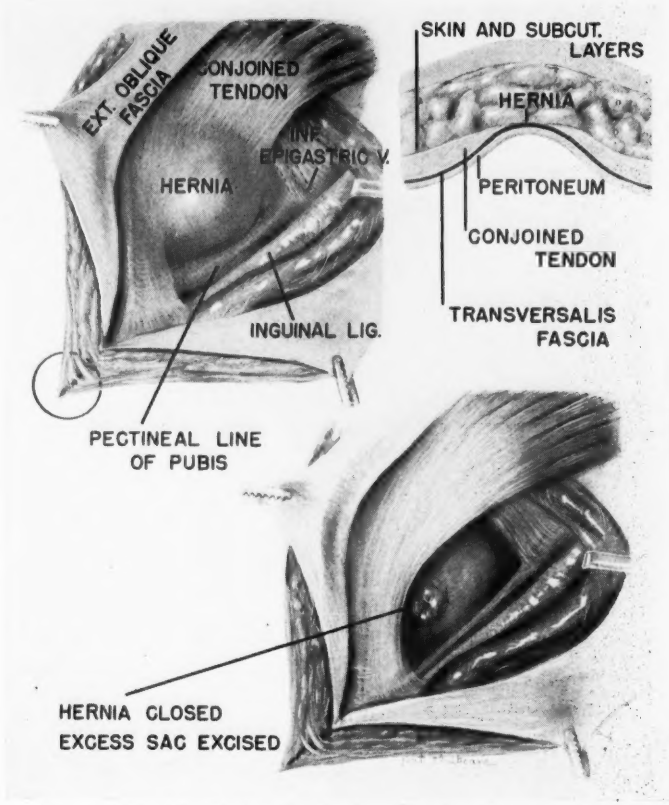

FIG. 2.—Drawing showing the development of a direct hernia and its relations to the pectineal line of the pubis. Note the defect through Hesselbach's triangle after the sac has been ligated.

A hernia may occur if there is a short attachment of the tendon along the pectineal line. This leaves a point of natural weakness just medial to the deep inferior epigastric vessels. After years of stress and strain the peritoneum and transversalis fascia may push forward through this weak spot. By continued stretching the conjoined tendon is worn away and a true direct hernia develops. By the same process an indirect hernia may gradually encroach upon and displace the attachment of the conjoined tendon and a combination direct and indirect hernia is formed. In both of these types the true transversalis fascia is stretched in front of the protruding peritoneum and may form part of the covering of the hernia. If the hernia becomes very large the transversalis may be destroyed over the dome of the sac. The majority of direct herniae result from prolonged stretching and thinning-out of that portion of the conjoined tendon extending from the edge of Gimbernat's ligament to the margin of the deep inferior epigastric vessels (Fig. 2). In this type the conjoined tendon may be completely separted from its attachment to the pubis. In other cases it may remain partly attached. As in the first type the transversalis fascia frequently is simply stretched in front of the peritoneum and forms one of the layers of the covering of the hernia. Occasionally a hernia develops through a longitudinal split in the conjoined tendon. Regardless of the mode of development, the important alteration in the anatomy is the disintegration of that part of the conjoined tendon that is normally attached to the pectineal line of the pubis.

If there is to be an anatomic repair of a direct hernia the operation must include the reconstruction of the damaged conjoined tendon and the restoration of its insertion into the pubis. Direct attempts to repair the damaged tendon and to reestablish its attachment fail except in the case of a very small defect. The hernia usually produces a wide gap in the abdominal wall between the intact portion of the tendon and the superior ramus of the pubis. The inelastic character of the tendon makes it impossible to reunite this structure to its normal place of insertion. The operations of Wölfler, Bloodgood, Halsted, Hotchkiss, Lusk, Berger, and Downes all aim to create a buffer in front of the defect in the floor of Hesselbach's triangle. None have materially reduced the recurrence rate of direct herniae.

In order to accomplish a reconstruction of the floor of Hesselbach's triangle a substitution of the destroyed structure must be made. In search for some suitable "substitute tissue" my attention was turned to the study of the ultimate fate of various types of autogenous grafts. From the excellent communications by MacEwen,[2] and Gallie and Robertson,[3] it would appear that an osteoperiosteal transplant applied to bone or periosteum becomes firmly united to those structures through the formation of fibrous tissue. Furthermore, when periosteum, bone and periosteum, or bone alone is transplanted into tissue where they are not normally found; or where there is no function for them to perform; they are replaced by dense fibrous tissue scar. In a series of experiments upon dogs I found that when free osteoperiosteal grafts were attached to the transverse processes of the spinal vertebrae and to the spinal

muscles they became absorbed within a few weeks. However, there had been left a thick, dense layer of fibrous tissue firmly united to the muscles and the transverse processes. It was also found that bone and periosteum was more fibrogenetic than periosteum alone. It seemed then that an osteoperiosteal transplant would answer the purpose for the "substitute tissue" with which to reconstruct the floor of the Hesselbach's triangle. Upon the basis of these observations and from the consideration of the pathologic anatomy of a direct inguinal hernia, the new principle which I now employ was developed. Since the preliminary report of the operation was published certain changes have been made. Therefore, I wish to describe in more detail the technic of the procedure in this communication.

Technic of the Operation.—The lower abdomen, including the inguinal region, is prepared in the usual manner. One leg is also prepared from the knee to the ankle. The sterile drapes are so placed that both fields are exposed. Since this operation entails two distinct procedures in two unrelated parts of the body teamwork is essential. The plan has been for one surgeon to devote his attention to the inguinal region while his assistant removes the osteoperiosteal graft from the anterior surface of the tibia. In order to follow the operation in steps the preparation of the graft will be described first.

The Osteoperiosteal Transplant.—An incision about eight inches in length, beginning at the level of the tibial tubercle, is made over the anterior surface of the tibia. The skin is reflected, thereby exposing the periosteum covering the broad anterior surface of the bone. The periosteum to be removed is outlined with the point of the scalpel. Its shape is somewhat like that of a tennis racquet, the oval part being the head piece and the handle forming the tail portion (Fig. 3). The size of the head piece is determined by the width of the defect produced by the hernia as it protrudes through the floor of Hesselbach's triangle. It must be wide enough to completely bridge this defect. It is cut from the enlarged upper end of the tibia. The tail piece is simply the prolonged tapering portion of the graft, and is about six inches in length. After the graft has been outlined on the tibia the periosteum is divided about its entire course (Fig. 3). The head piece is elevated with a chisel by cutting thin slivers of bone from the tibia, and leaving them attached to the periosteum. When the tail portion is reached the periosteum is simply elevated from its bed. The graft then consists of an oval shaped head piece of periosteum covered with slivers of bone and a thin tail portion of free periosteum. After the graft has been removed the incision is closed by bringing the skin edges in apposition with interrupted silk sutures. It is not necessary to attempt to close the periosteal bed.

Technic of Hernioplasty.—An oblique incision is made over the inguinal canal. The external oblique fascia is divided and the cord and hernia are exposed. The cord is retracted laterally, and the exact nature of the hernia is determined. The size of the defect in the floor of Hesselbach's triangle is noted. The sac is then picked up in forceps and incised at its apex. A purse-string suture is placed around its base and the excess portion is excised. The

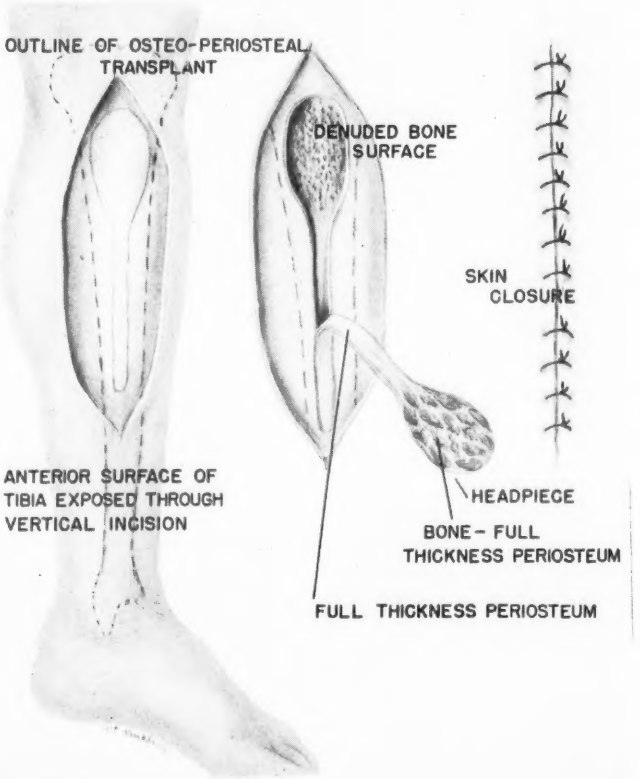

FIG. 3.—Drawings showing the removal of osteoperiosteal graft from the tibia. Note that the head piece of the graft contains bone and full-thickness periosteum. The tail piece is only periosteum.

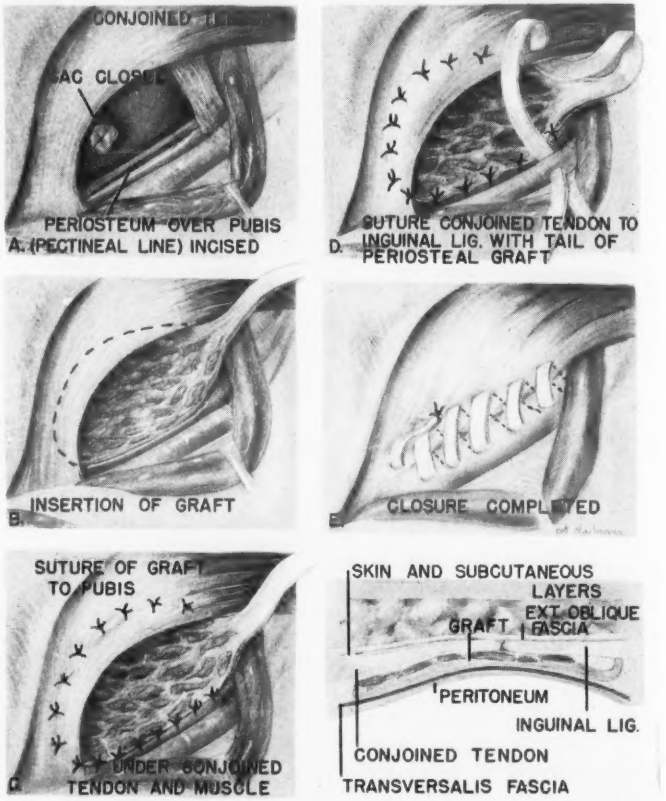

FIG. 4.—Drawings showing technic of repair of the anatomic defect in the conjoined tendon in a direct hernia. Note that the bone surface of the graft is turned outward. Note also the use of the tail piece as a fascial suture.

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inferior epigastric vessels are identified. The conjoined tendon is freed from all areola tissue and fat along its free edge. The ligated sac is then pushed away from the pubis by gentle finger dissection. The periosteum just posteriorly to the pectineal line of the pubis is incised from the lateral edge of the Gimbernat's ligament to the margin of the inferior epigastric vessels (Fig. 4). If these vessels seem to interfere with the closure they are ligated and divided. The field is now ready for the reception of the graft. The head piece of the transplant, with bone surface upward, is placed in the gap between the pectineal line of the pubis and the conjoined tendon (Fig. 4). The inferior margin of the transplant is sutured into the slit in the periosteum of the pubis with interrupted silk sutures. The superior margin of the transplant is placed posterior to the conjoined tendon and sutured to that structure with interrupted silk sutures. These sutures extend from the angle at Gimbernat's ligament to the edge of the tail of the transplant. This portion of the transplant is now utilized to approximate the free margin of the inferior border of the conjoined tendon to the inguinal ligament after the manner used for the Gallie-type of fascial suture. The first suture is passed through conjoined muscle, just above the beginning of the tendon, then down through the inguinal ligament (Fig. 4). From this point the suture is woven back-and-forth from the conjoined tendon to the inguinal ligament until these structures are approximated from the inferior epigastric vessels to Gimbernat's ligament. A final silk suture is used to fix the end of the transplant into the tendon. The cut edges of the external oblique fascia are closed behind the cord. The cord is now placed in its new bed and the subcutaneous tissues and skin closed in the usual manner.

The first operation using this new principle was performed in February, 1937, at Charity Hospital, New Orleans. During the following 16 months, 20 additional such repairs were performed at the same institution. All of the cases were carefully followed—14 for a period of six months to one year. There was not a single recurrence. In July, 1938, I came to Gallinger Municipal Hospital, Washington, D. C., and have lost contact with the Charity Hospital cases. My associates and I have performed the same operation upon 64 patients at the Gallinger Hospital, and have succeeded in following all of these by personal observation. In 34 cases the follow-up period ranges from one year to two and one-half years. Six months to one year has elapsed since operation upon 20 additional patients. Including the Charity Hospital group, there are now 68 cases in which the follow-up period has been at least six months. In the entire series of 85 cases there has been only one recurrence. This failure was the direct result of a gross infection of the wound. It occurred early in the postoperative period and the entire graft was eventually extruded.

The postoperative care of these cases has been the same as for all inguinal herniae. The patient is allowed out of bed on the fourteenth day. He may resume his usual occupation eight weeks following his operation. The healing process seems to be just as rapid as that following the simple Bassini operation. There is one great difference noted in the osteoperiosteal repairs—the recon-

structed wall is thicker and more rigid. Even in the cases with the longest follow-up period there still remains a strong inguinal wall, which is firmly united to the pubis. This obvious building-up of a new fibrous wall has not been attended by any discoverable ill effects. Since the cord is transplanted outside the fascial layers there has been no damage to this structure. The osseous portion of the transplant is gradually absorbed. It has been possible to follow this process by means of repeated roentgenologic studies. The first roentgenogram, made during the early days of the convalescent period, has shown the outline of the bone in the graft merging into the denser shadow of the pubis along the line of the attachment. Roentgenograms made six weeks later failed to show this shadow in some, but it was still present in others. In all the cases studied there has apparently been a complete absorption of the bone within three months.

There have been three wound infections, two of these involving all layers, the other being only superficial. In one of the grossly infected wounds, the slivers of bone contained in the graft were gradually sloughed, but there has been no recurrence of the hernia two years after the original repair. In the other grossly infected wound the entire graft was extruded within two weeks after the repair, and there was an immediate recurrence of the hernia. The leg wound has healed in all cases by primary union. There has been no evidence of any damage to the tibia. In one case an hematoma developed under the skin and had to be evacuated, but no serious consequences followed.

Although the number of cases included in this report is small, and the follow-up period inadequate, certain conclusions can be drawn. A clear understanding of the pathologic anatomy of a direct hernia is essential. The operation does not require any special equipment or any unusual postoperative care. The removal of the osteoperiosteal graft from the tibia has not caused any disability or prolonged the convalescent period. The results of the operations usually employed for direct hernia show that about one-half of the recurrences develop within six months. In the present series, 68 consecutive cases repaired with the osteoperiosteal transplant have remained intact over a six-month period. This seems to indicate that the anatomic defect has been successfully bridged. The permanency of the repair can be determined only by a much longer follow-up study.

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UNUSUAL VISCERA IN INDIRECT INGUINAL HERNIAE

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THE PRESENCE OF AN OVARY in the sac of an inguinal hernia is not common, most surgeons of large experience having encountered it one or more times. However, the presence of the uterus in an indirect inguinal hernia is rare. The earliest report of an inguinal hysterocele was made by Nicholas Pol, in 1531. While it is not stated, this was undoubtedly a direct type of hernia. Senertus and Hildarus, in 1610, reported a case which they claimed was identical to the one described by Pol. Another case described by these authors, and which was attributed to Doringuis, is variously classified as crural or inguinal. Study of these early reports shows that considerable confusion existed both as to the authenticity and exact nature of these cases.

Watson collected from the literature 30 cases of hysterocele, and 61 cases of hernia of the nonpregnant uterus, a total of 91; 219 cases of hernia of the adnexa, 80 cases of tubal hernia, and 181 cases of the ovary alone.

While it is impossible to give the exact percentage of incidence of this complication, it can be readily seen that it would be very small considering the number of herniotomies performed.

Few of the cases reported are accurately described as to whether they are direct or indirect inguinal herniae. For example, Jobson, in 1904, and Andrews, in 1906, reported hernia of the uterus which was apparently of the direct type. Makkas, in 1910, reported bilateral uterine hernia of a bicornate uterus, and here again a direct hernia existed. Only one author, Ludington, in 1920, definitely recorded a case of the presence of the uterus in an indirect inguinal hernia in a child, age 19 months.

So far as the presence of ovaries and tubes in the sac of an inguinal hernia is concerned, we find in the literature numerous references, the earliest appearing in the writings of Soranus, of Ephesus, about A. D. 97. McNealy, in 1914, reported a case of strangulated tubo-ovarian hernia in an infant, and, in 1920, a sliding hernia of the fallopian tube. Deutschman, in 1923, reported a case of congenital absence of the vagina associated with bilateral hernia of a uterus bicornate, tubes and ovaries. In 1928, Sarnoff reported a case of direct hernia of the uterus and tubes through the inguinal canal. Further review of the literature shows a case of direct inguinal hernia containing the ovary, tube and horn of the uterus, reported by Hilarowicz, in 1928. In 1938, Reinberger published an interesting study of the human ovo-testis associated with a congenitally bisected uterus herniated into the inguinal canal.

While these cases are rarely encountered, it seems worth while to report them and to call attention, again, to such complications arising in the surgical problems of hernia.

CASE REPORTS

Case 1.—A white woman, age 30, married, by occupation a professional acrobatic dancer, was admitted to Wesley Memorial Hospital, February 9, 1941, complaining of a lump in the right groin which appeared after a fall at the age of eight years. The lump could be reduced until eight years ago, since which time it has persisted in its

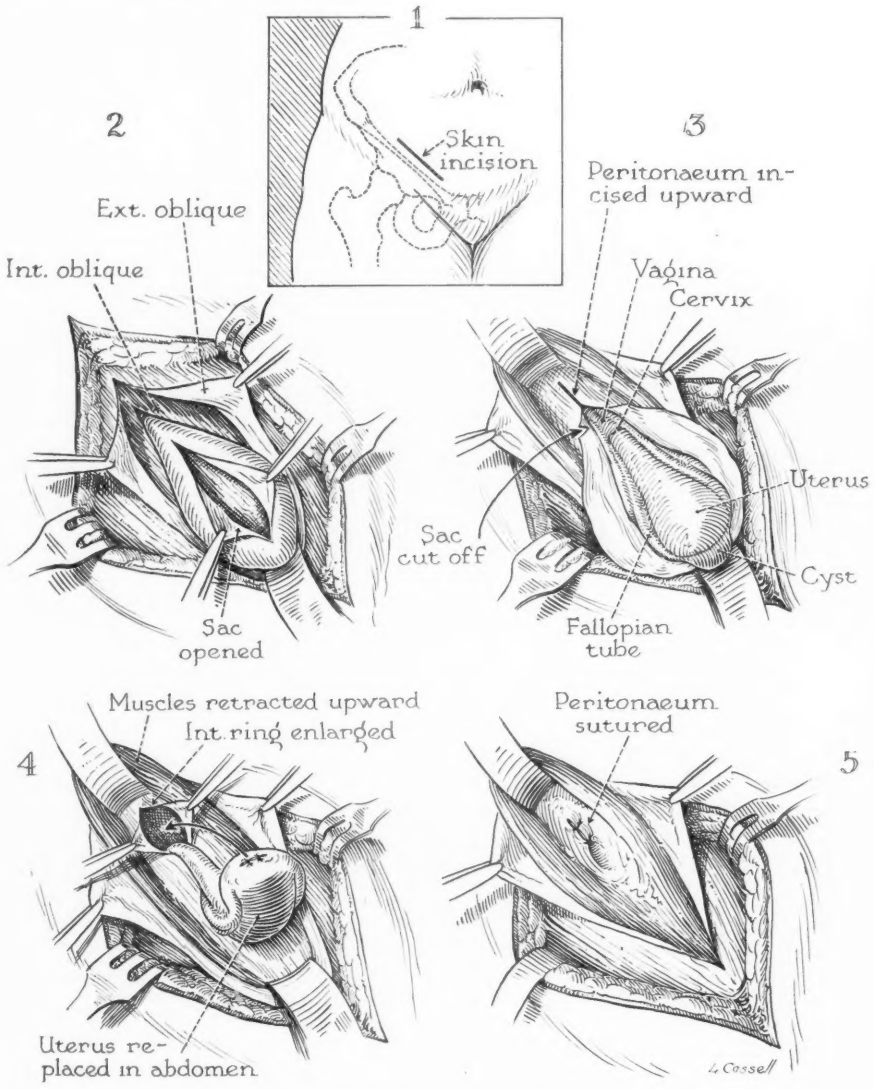

FIG. 1.—Sac of the indirect inguinal hernia opened anteriorly, showing rudimentary uterus.

present location. A left inguinal herniorrhaphy had been performed at the age of six. The remainder of her history was essentially negative except that she had never menstruated, and that there was a familial tendency toward hernia, as evidenced by a history of hernia in her grandfather, mother, sister and nephew.

Physical Examination.—This revealed entirely negative findings throughout except for the presence in the right groin of a mass the size of a hen's egg, which presented

its tip at the external ring, giving rise to an impulse on coughing. Pelvic examination showed the absence of the cervix; the uterus could not be palpated. All secondary sex characteristics were normal.

Operation.—Avertin and cyclopropane anesthesia: After exposure of the sac, which was found coming through the internal inguinal ring, it was incised and found to contain a rudimentary uterus which had undergone slight cystic degeneration at the fundus (Fig. 1). The cervix could be felt at the base of the sac near the internal ring, although it was necessary, of course, to palpate it through the thickness of the vaginal walls and urinary bladder. The adnexa were not present in the sac. The opening into the abdomen at the neck of the sac would only permit the insertion of the tip of the index finger and it was, therefore, impossible to replace the uterus without enlarging the opening. Due to the failure to find the uterus on bimanual examination prior to operation, the patient had requested that should any abnormal contents of the sac be present she did not wish their removal unless absolutely necessary. The opening into the abdomen was, therefore, enlarged by retracting the muscles upward at the internal ring and incising the peritoneum in the same direction (Fig. 1-3). The uterus was then replaced and the usual repair employed after closing the peritoneum.

The patient made an uneventful recovery except for a minor urinary complication, and returned to her work one month after operation.

COMMENT.—This case brings up the possibility of the severance of the left round ligament at the time of the first herniotomy at the age of six, since the right hernia appeared two years later, perhaps brought on by her acrobatic activities which she stated commenced in childhood.

Case 2.—A white woman, age 29, housewife, married, was admitted to Wesley Memorial Hospital, April 10, 1941, complaining of a lump in each groin present since the age of two. These lumps appeared after a fall, and her parents at once applied a truss which she wore until the age of nine. During the last two years she had noticed a pulling sensation associated with some soreness over the swellings, which had first appeared following some corrective exercises.

Her past history was essentially negative except that she had never menstruated. On pelvic examination by a gynecologist some years previously, she was advised that the uterus could not be found.

Physical Examination.—This revealed negative findings throughout, except for the presence of two irreducible masses, bilaterally, at the external rings. Pelvic examination revealed the absence of the cervix in the vagina, and the body of the uterus could not be palpated bimanually. All secondary sex characteristics were normal.

Operation.—Cyclopropane anesthesia: Exploration of the sac revealed it coming through the internal ring of the left side. Upon opening it, it was found to contain a structure which resembled an undifferentiated gonad the size of a normal testis, accompanied by its vascular supply (Fig. 2). The processus vaginalis of this structure was opened, the edges everted and sewed posteriorly to the organ (bottle operation). No communication could be found into the abdominal cavity. Since it is believed by some authorities that extirpation of such structures produces mental changes, it was decided to return the gonad to the abdomen after a biopsy specimen was obtained. Accordingly, the muscles were retracted upward at the internal ring and the peritoneum was incised in the same direction, down to the proximal end of the incision in the processus vaginalis. Through this opening an effort was made to locate the uterus, but it could not be found, although the opening was not sufficiently large to be certain. The gonad was then replaced in the abdomen and, after closure of the peritoneum, the usual repair of the hernia was carried out. An incision was then made on the right side. The findings were the same as on the left side, and, consequently, the same technic was employed.

Pathologic Examination.—Microscopic: Dr. E. R. Strauser. "The biopsy specimen consists entirely of fibrous tissue with the exception of some tubular structures which are probably of testicular origin. This cannot be definitely stated because they also resemble embryonic remnants, which are often found about the tubes. I do not believe they belong definitely to either the male or female genitalia."

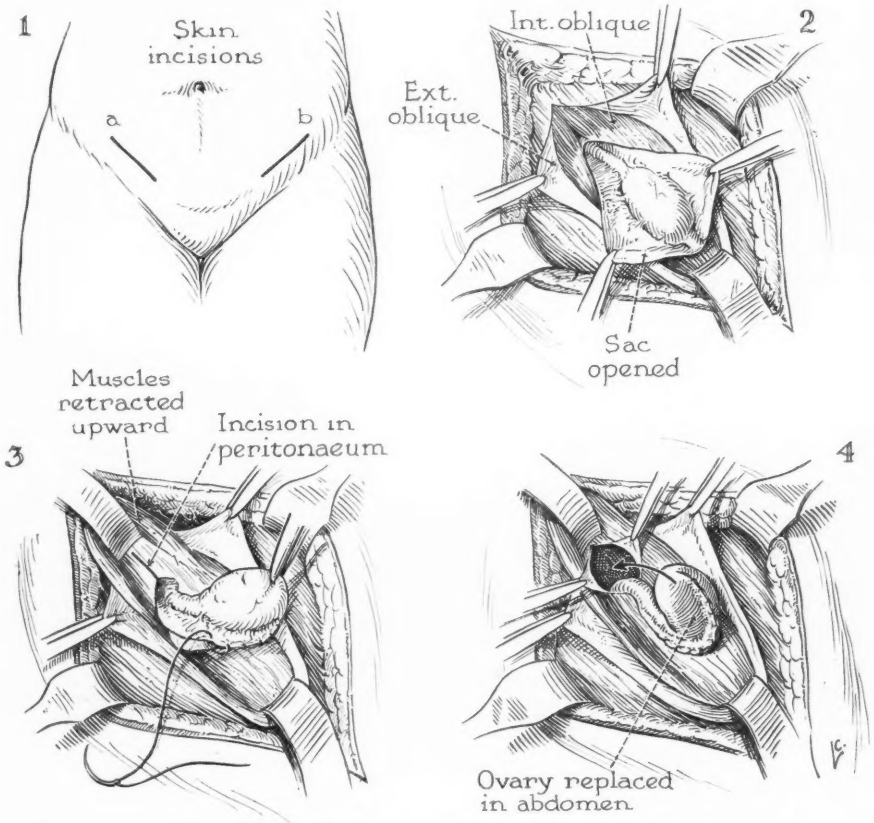

FIG. 2.—Sac of the right indirect inguinal hernia opened anteriorly, showing the gonad lying midway along the inguinal canal. A similar condition was found on the left side.

CONCLUSIONS

Two cases are reported, one containing the uterus in the sac of an indirect inguinal hernia, and the other, gonadal structures on each side of a bilateral indirect inguinal hernia.

Such findings are infrequent, according to the literature, and are reported here for the purpose of calling attention to the complications that may arise in so common a condition as inguinal hernia.

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PRODUCTION OF A THROMBOTIC BARRIER IN THE TREATMENT OF VARICOSE VEINS*

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THIS PAPER is concerned with the description of a simple, nonoperative method for limiting the chemical thrombophlebitis following retrograde injection of the internal saphenous vein during high ligation to the thigh portion of that vein.

Retrograde injection of the internal saphenous vein during ligation was first practiced by Tavel[15] in 1904. Moszcovitz,[11] in 1927, reemphasized its advantages. However, the level of ligation described by both these writers was considerably lower than that at which it is performed today. In 1934, Edwards,[3] in an excellent anatomic study, graphically illustrated how the point of ligation of the internal saphenous vein has gradually moved upward, so that today it is generally accepted that ligation of this vein should be done at the femoral junction with separate ligation of all available tributaries. In this country, Faxon,[4] in 1934, was first to describe the advantages of retrograde injection. Since that time, this method of treatment of varicosities involving the internal or great saphenous vein, that is, high ligation at the femoral junction, with separate interruption of all tributaries entering into it, and the injection of a sclerosing solution distally, has come to be accepted as the most efficient mode of treatment in cases where there is evidence of incompetence of the valves of this vein, as demonstrated by a positive Trendelenberg test.

The Thigh Portion of the Internal Saphenous Vein.—It is interesting to note that early in the injection treatment of varicose veins the failure to obliterate the thigh portion of the internal saphenous vein was recognized as a primary, if not the most important, cause of recurrence. DeTakats,[2] in 1930, and McPheeters,[8] in 1931, stated that the existence of a column of blood in the unobliterated thigh portion exerts pressure upon the thrombosed varicosities in the leg and so contributes to their recurrence. M. Pheeters,[9] in order to eliminate the patent high portion, recommended injection of the internal saphenous vein from ankle to thigh in one sitting.

It would seem that there are three main factors involved in the recurrence of varicosities that have been thrombosed by injection without previous ligation. The first is the fact that all thrombi have a physiologic tendency to recanalize unless the irritating agent has caused a very severe inflammatory reaction involving the entire vein wall. The second factor has recently been well illustrated by the work of Adams,[1] who showed that the pure gravity effect of a column of blood from the right auricle to a point of measurement in the mid-calf produces a pressure of 64 to 88 mm. of mercury in the standing position in patients with varicose veins, regardless

* Presented before the Chicago Surgical Society, May 1, 1942.

of whether the valves are competent or not. He pointed out that the presence of functioning valves has no effect on this gravity pressure since the fact that this venous system is full of fluid (blood) fulfils the requirements of Pascal's law. The third factor is the effect of straining on this standing or gravity pressure. Adams found that straining caused the pressures mentioned above to be increased by from 12 to 136 mm. of mercury resulting in a pressure of 224 mm. in one of his cases, a figure far above the normal systolic arterial pressure. The increase in pressure produced by strain he has shown to be particularly high in individuals with incompetent saphenous valves. After high ligation, the straining pressures were reduced by from 20 to 110 mm. of mercury.

The work of Adams, thus, very well illustrates the value of ligation. However, it is evident that ligation alone, without a simultaneous obliteration of the thigh portion of the internal saphenous vein, would still leave a column of blood exerting a pure gravity pressure on the leg veins. Adams work, thus, corroborates the rationale for retrograde injection since this procedure, when successful, insures the obliteration of the thigh portion of the internal saphenous vein.

Untoward Reactions Following Retrograde Injection.—Retrograde injection of a sclerosing solution at the time of ligation is now quite generally accepted as the method of choice in the treatment of varicose veins involving the internal saphenous vein in cases where its valves are shown to be incompetent by means of the Trendelenberg test.[5, 6, 7, 10, 12, 13, 14, 16] However, the writer has frequently witnessed very marked reactions following this procedure. In his experience this is particularly true in patients with extensive varicosities in whom the ensuing chemical thrombophlebitis and periphlebitis, involving as it does the whole internal saphenous tree, causes disability for a week or longer.

ILLUSTRATIVE CASE REPORT

Mrs. R. C., age 52, on examination, May 23, 1940, was found to have large varicosities involving the left internal saphenous vein. On the right side, there was moderate involvement of the external or small saphenous vein. The Trendelenberg test revealed a marked reflux phenomenon, and the Perthes test indicated normal patency of the deep veins. Point seventy-five cubic centimeters of sodium morrhuate was injected into one of the veins of the left leg, to make certain that the patient was not sensitized to this solution. Two days later, a high ligation of the left internal saphenous vein was performed, and four cubic centimeters of sodium morrhuate was injected distally. That evening, the patient complained of severe pain and discomfort. When seen the following morning, there was considerable redness, moderate induration, and marked tenderness all along the course of the saphenous vein from ankle to groin. The oral temperature was 104° F. This patient remained in bed for eight days. For the first six days, there was a low-grade fever ranging from 99° to 100.6° F. Codeine and hypnotics had to be administered to keep the patient in a degree of comfort.

It is obvious that this type of reaction tends to defeat the ambulatory nature of the injection treatment. Lowenberg[7] has called attention to similar experiences. In 1937, he stated that in order to obviate the considerable

disability resulting from extensive swelling, pain, etc., he regularly ligated the saphenous vein at the knee prior to high ligation and retrograde injection, and thus limited the thrombophlebitic reaction to its thigh portion.

Retrograde injection of the internal saphenous vein during high ligation has been practiced in this clinic since 1936. The writer has always felt that the problem of eliminating varicosities of the internal saphenous vein was essentially a problem of sclerosing its thigh portion. About 80 per cent

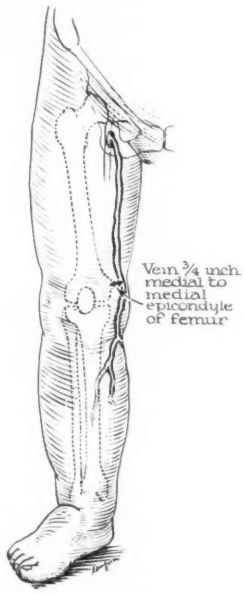

FIG. 1.—Drawing illustrating the relationship of the internal saphenous vein to the medial epicondyle of the femur.

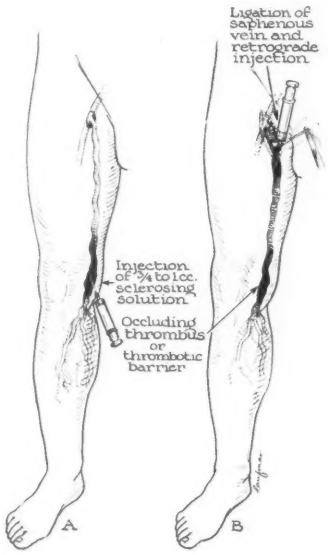

FIG. 2.—Drawings showing the proper location of the occluding thrombus or thrombotic barrier. The optimum location is slightly above an imaginary horizontal line bisecting the patella.

of cases of varicosities occur in women. In them, the thigh portion of the internal saphenous, unlike the leg portion, is covered by a thick layer of fatty tissue, making palpation of this vein in the thigh, for purposes of injection, often quite difficult. This is particularly true after simple ligation. In the experience of the writer, severe reactions following retrograde injection, as illustrated by the case report, and described by Lowenberg, occur in one-half to two-thirds of cases with severe varicosities. In the past year, the following method has been used to limit the chemical thrombophlebitis to the thigh portion of the internal saphenous vein in all cases where high ligation with retrograde injection is planned.

METHOD OF PRODUCTION OF THROMBOTIC BARRIER

The internal saphenous vein at the level of the knee is palpated. At this point, the vein will be found about three-quarters of an inch medial to the

medial epicondyle of the femur (Fig. 1). Its palpation is facilitated by the fact that the layer of fat at this level is much thinner than on the thigh proper. The vein is injected with 1 cc. of sclerosing solution (sodium morrhuate). The patient is asked to return in three or four days. If no thrombosis has occurred, the vein is reinjected with 1.5 to 2 cc. of solution, depending upon the caliber of the vessel. Any palpable tributary in the region of the knee is similarly treated. Ligation and retrograde injection is performed when a firm thrombus has resulted. This usually takes ten to 14 days. In this way, an occluding thrombus, or thrombotic barrier, is created which prevents the sclerosing solution injected into the internal saphenous vein at operation from passing below the level of the knee (Fig. 2).

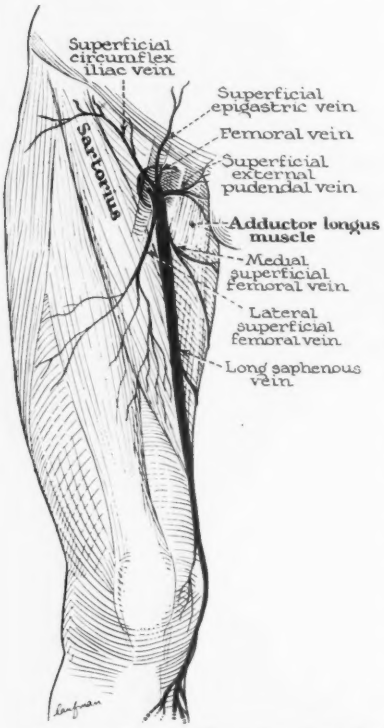

FIG. 3.—Drawing illustrating the relationship of the internal saphenous vein in the fossa ovalis to the easily palpable inserting portion of the adductor longus muscle. The vein can be felt one-half to one inch lateral to this landmark in the great majority of cases. Note the relationships of the five main tributaries. The upper two, and occasionally even the superficial femoral veins, may join the femoral instead of the internal saphenous vein.

The experience with 95 cases in whom the production of a thrombotic barrier preliminary to ligation was attempted, has been as follows: In 62 cases (65 per cent), firm thrombosis of the saphenous vein at the knee was obtained after the first injection. In 14 cases (15 per cent), two injections were necessary, while in 12 instances (13 per cent), three or four injections were required. In seven cases (7 per cent), no thrombosis was obtained after four injections and ligation with retrograde injection was performed in the usual manner.

Method for Locating the Internal Saphenous Vein in the Fossa Ovalis.—Many suggestions have been made for locating the internal saphenous vein in the fossa ovalis. Briefly, the most common procedures employed are the following: Estimating the location of the vein by measuring one inch medial to the femoral artery, which is located by its pulsation; palpating the transmitted impulse over the vein at or near the fossa ovalis following percussion of the vein in the region of the knee or lower thigh; using the pubic tubercle as a fixed point and measuring an average of 1.5 cm. below and 4 cm. lateral to it, in order to locate the fossa ovalis.

For the past six years, the following method has been employed for locating the internal saphenous vein in the fossa ovalis preliminary to ligation

and for the performance of the Trendelenberg test. With the patient in the standing position, the examining fingers are placed over the thick, firm, adductor longus muscle at its tendinous insertion and are then moved one-half to one inch laterally, where the vein will be felt as a soft, yielding tube (Fig. 3). It is easy to palpate the vein by the use of this method even in the most obese patient because of the fact that the layer of fat in this region, as over the knee, is relatively thin and also because the vein, in the varicose state, is almost invariably dilated. The course of the vein is then traced on the skin with a dye.

Operative Technic.—The operative procedure followed is essentially like that described by Faxon, and other writers. A short transverse incision, perpendicular to the dye mark, is made about one-half inch below the inguinal fold. The superficial fascia is then incised in a direction parallel with the skin incision. The vein is separated from the fatty and areolar tissues surrounding it and is then ligated high with No. 00 chromic catgut. This first ligature serves for traction downwards to aid the exposure of the two uppermost tributaries—the superficial epigastric and superficial circumflex iliac veins. These, if tributaries of the internal saphenous instead of the femoral vein, as they sometimes are, are separately ligated and severed. Another ligature is then placed on the saphenous vein above their point of juncture. The superficial external pudendal vein is similarly treated. The lateral and medial superficial femoral veins, if present, are not ligated since that would prevent the sclerosing solution from entering them. Three to four cubic centimeters of sodium morrhuate are then injected into the saphenous vein distally. A No. 16-gauge needle, previously bent to an angle of 120°, to facilitate entrance into the vein, is used for this purpose. The vein is then ligated again about one inch below the point of injection and the segment of vein between the highest proximal and the distal ligature is excised.

Within 24 to 48 hours, the thigh portion of the internal saphenous vein becomes thrombosed. Often, in over one-half of the cases, the ensuing reaction is accompanied by considerable periphlebitis. However, none of these patients have been incapacitated for longer than a day. The injection of the patent leg varicosities is begun two weeks after ligation and is done at weekly intervals.

COMMENT.—The comfort and the ability of these patients to engage in their usual pursuits has been in direct contrast to the discomfort and disability suffered by most patients who have been subjected to ligation and retrograde injection without the creation of a barrier at the knee. Possibly, the reason for the absence of disabling symptoms is to be explained by two factors: First, the limitation of the thrombophlebitic and periphlebitic process to a smaller surface; and second, the absence of inflammation in lower region of the knee and leg, resulting in painless locomotion.

SUMMARY

1. The combined ligation-retrograde injection treatment of varicose veins often causes considerable disability and discomfort in patients with marked involvement of the internal saphenous vein.

2. A method is described whereby a thrombotic barrier, or occluding thrombus, is created in the internal saphenous vein in the region of the knee for the purpose of limiting the thrombophlebitic process to this level. This results in practically no disability to the patient. An experience with 60 patients is described.

3. A simple method for locating the internal saphenous vein in the fossa ovalis, preliminary to ligation or to performance of the Trendelenberg test, is described.

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HEMISECTION OF THE MANDIBLE FOR RECURRENT ADAMANTINOMA

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SINCE there have been a number of recent excellent reviews of adamantinoma, there is little indication for repetition. Hence, it is not our purpose to approach the subject from such a standpoint but merely to report two cases which were treated by hemisection of the mandible. Both cases had been treated less radically, on one or several occasions, with subsequent recurrences. It is of interest that the mutilation is less than might be anticipated even without dental prosthesis, and the result from a functional standpoint is gratifying.

Case 1.—N. E., colored, female, age 29, was admitted to the Lakeside Hospital, June 27, 1934, complaining of swelling of the left side of the lower jaw, which had had its onset shortly after the extraction of a tooth some two years previously. Growth during the preceding nine months had been exceedingly rapid. During recent months the patient had had difficulty in separating the teeth more than one-half centimeter. The tumor mass was of the dimensions indicated in the photographs (PLATE I a and b), and in general was quite firm, although just below the zygoma it had a less firm character. The patient had a rather marked secondary anemia which could not be accounted for. On three different occasions partial resections and curettements had been carried out with ensuing recurrences.

Operation.—October 31, 1935: Without preliminary ligation of the external carotid being carried out, a hemisection of the mandible was performed under intranasal catheter anesthesia. The mandible was divided through the symphysis and disarticulated at the temporomandibular joint. The mouth was opened into and an area of mucosa was removed with the jaw where it was intimately attached to the tumor. Considerable respiratory difficulty developed when the incision was extended posteriorly to the pharyngeal group of muscles; however, this was entirely corrected by the insertion of an airway. Closed intratracheal anesthesia might be useful; however, with reasonable care the above type of anesthesia proved quite satisfactory. The mucosa and submucosa were closed and a drain was inserted in the subcutaneous tissues at the posterior portion of the incision. The wound healed without a fistula in about five weeks. *Histologic Report:* Adamantinoma.

One week postoperatively the teeth were wired to avoid deformity; the wire was removed after five weeks (Case 2 was not wired). The subsequent course was satisfactory and there was no evidence of recurrence of the lesion in the mandible. However, about six months later an obviously cystic swelling was noted in the left temporal fossa which gradually increased in size (dotted circle in PLATE I b). It was definitely in the temporal muscle and it was thought it represented an extension from the coronoid process or actual retraction of neoplastic tissues with the temporal insertion at the time of the resection of the mandible. On May 1, 1936, the left temporal fossa was exposed through an incision above the hair line; the zygoma was resected its full length for exposure and the cystic lesion was entirely removed. The premise previously suggested as to its origin was consistent with the findings. Examination in March, 1942, shows there to be a local recurrence in the temporal fossa but none in the region of the mandible.

PLATE I.—Case 11: a, b, and c—before operation. d, e, and f—after operation. The dotted circle in b shows the cystic swelling in left temporal fossa.

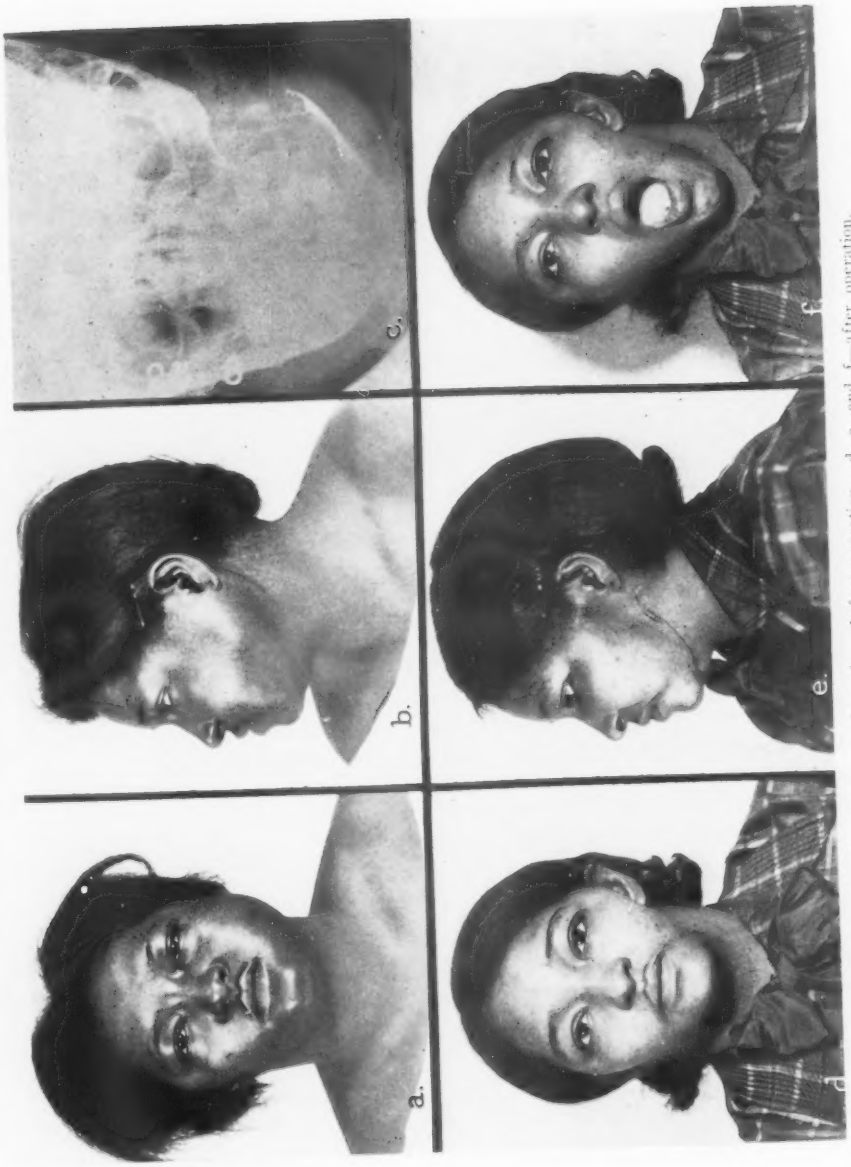

PLATE II.—CASE 2: a, b, and c—before operation. d, e, and f—after operation.

Case 2.—L. J., colored, female, age 30, was admitted to Lakeside Hospital, September 30, 1932, complaining of swelling of the left side of the jaw which she had first noted some 12 years previously following the extraction of teeth. Increase in size had been gradual and not painful. The patient recently had noted cystic projection within the mouth. Examination revealed a swelling of the dimensions indicated in the photograph (PLATE II a and b); this swelling only slightly impaired the ability to open the mouth. To palpation exteriorly it was neither bony hard nor definitely cystic; however, there were several projections into the mouth which were definitely cystic. On October 4, 1932 partial resection and curettement were carried out. *Histologic Report:* Dentigerous cyst (follicular).

The subsequent course was satisfactory for several years; however, cystic areas reappeared in the mouth, and roentgenograms revealed involvement of the mandible up to the temporomandibular joint. On January 20, 1937, hemisection of the mandible was carried out as described in the preceding case, although in this instance the resection was between the canine and lateral incisors. Examination of the resected specimen revealed the expansile effect of the lesion which was further manifested by the upward migration of the coronoid process (Fig. 1). There might also have been a degree of

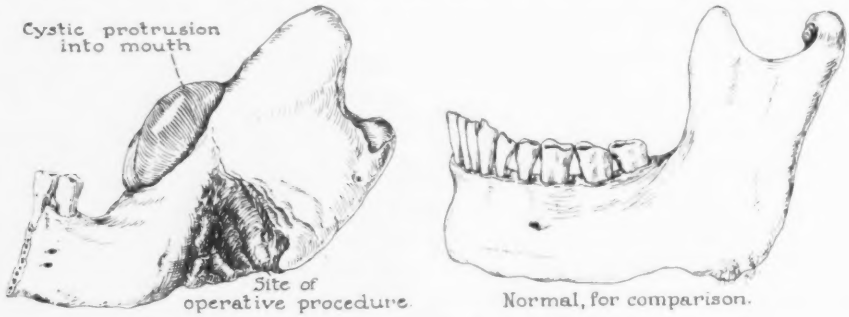

FIG. 1.—Case 2: Resected half of mandible.

rotation as a result of the previous local resection of the mandible which might explain the apparent migration of the coronoid process upward. However, in other instances of expansile lesions we have noted apparent upward migration of the coronoid. *Histologic Report:* Cystic adamantinoma.

The change in the pathologic diagnosis cannot be considered too significant in view of the similarity, microscopically, of the original lesion and the more cystic types of adamantinoma. The subsequent clinical course was satisfactory and there have been no recurrences to date.

While dental prosthesis might improve the cosmetic result, it was not employed in either of these cases. However, since the deformity is manifest by a flattening extending up to the level of the zygoma, this could not be entirely corrected by any form of dental appliance. The occlusion of the teeth of the remaining portion of the mandible is satisfactory and from a functional standpoint the patients can masticate quite well. The excellent muscle power may be demonstrated by noting the difficulty associated with the withdrawal of a tongue blade inserted between the teeth. Except for the loss of masticating surface there has apparently been little impairment of masticating efficiency of the remaining jaw. At no time have the patients complained of pain in the remaining temporomandibular joint as might be anticipated because of abnormal distribution of stress and strain. While from

the cosmetic standpoint a dental prosthetic appliance might be indicated, it is believed that from a functional standpoint it might place too much strain on the remaining temporomandibular joint, unless a fulcrum were established in the upper jaw on the resected side.

In the first instance resection was, of necessity, carried out in the midline, and if any deviation of the chin occurs it is away from the side of resection. In the second case it was practical to resect between the incisors and canine teeth, and it will be noted that there is slight deviation toward the side of resection. Although the teeth were not wired postoperatively in the second case, we are inclined to believe that the discrepancy in deviation is due to muscle pull, *i.e.*, the nature of the muscle attachments remaining (largely the preservation of the origin of anterior belly of the left digastric). This may well explain the observed tendency to more marked deviations in unilateral resections of much less magnitude than hemisection.

Our limited experience would lead us to agree that the lesion should be widely excised, and if it is of any extent that hemisection should be given consideration. The mutilation incident to hemisection may be less than anticipated and from the functional standpoint largely represents a diminution of masticating surface. Kimm and Baranoff[5] report a series of 26 cases of adamantinoma, in 13 of which they carried out hemisection of the mandible.

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SO-CALLED DISLOCATION OF THE LOWER END OF THE ULNA

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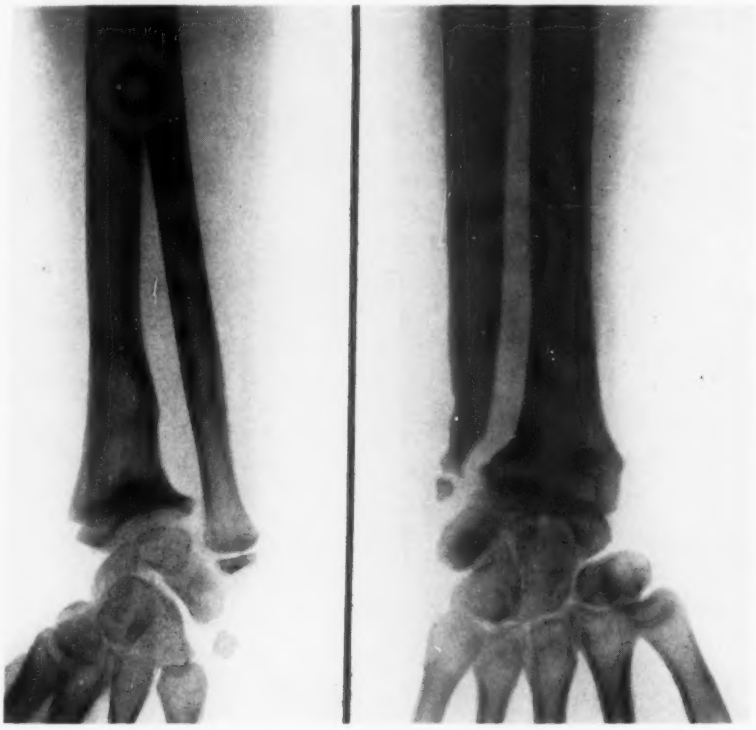
"DISLOCATION of the lower end of the ulna" has been described as occurring after Colles fracture, in Madelung's deformity, in arthritis of the inferior radio-ulnar joint, as well as in a variety of other fundamentally dissimilar affections. Though the very diversity of the circumstances under which these so-called dislocations have been discovered should, in itself, have cast some doubt upon either the validity of the diagnosis or the unique nature of the underlying pathology, this has not been the case. Largely, it appears that the diagnosis depends upon the fact that dorsal prominence of the ulnar head, especially on pronation of the forearm, is a common and distinguishing feature of these different conditions. Yet when the roentgenograms of individual cases are studied closely, any conclusion justifying such a diagnosis seems to be open to very serious question. In one, there may be an abnormal disproportion in the length of the radius and ulna. In another, there may be a diastasis at the inferior radio-ulnar joint, without any change in the relative lengths of the bones. In a third, the most striking feature of the roentgenogram may be an axial malalignment of one or both of the bones, while in still another no bony abnormality whatsoever may be present.

The observations of these varied roentgenographic appearances becomes more significant when the effort is made to understand just what is meant by "dislocation of the inferior end of the ulna." If the term is employed in its usual connotation, it should imply a displacement of the inferior end of the ulna in relation to a more proximally located joint surface. Manifestly, this cannot be intended, since it does include the conditions under discussion. But even if its meaning were extended, so as to embrace displacements in reference to more peripherally located surfaces, the term would still describe no clearly definable anatomic concept. Because of the continuity of the ulnar shaft and the solid inclusion of its upper end in the elbow joint, dislocation of the lower end cannot occur, except as the result of fracture of the shaft. In fact, it seems there is no such condition as "dislocation of the lower end of the ulna." The term is a misnomer and is misleading, because it is not the ulna which dislocates in relation to the radiocarpal mass, but, on the contrary, the latter which becomes displaced in respect to the stationary ulnar head.

Since, as will be seen, prominence of the lower end of the ulna is typical of a numer of different affections, a more accurate designation, based upon their common symptomatology, appears to be desirable. In general, the whole group is characterized by weakness of the wrist, pain or tenderness on pressure, a clicking sensation on rotation of the forearm, and an abnormal prominence of the ulnar head. By analogy with the terminology used in

describing many injuries to the knee as "internal derangements," it seems reasonable to suggest a similar designation, "derangement of the wrist," for the disabilities here reviewed. This difference in terminology is of more than academic significance, because of the therapeutic consequences which each of the two concepts entails. The one leads to a search for and a



A B

FIG. 1.—Case 1: (A) The site of the earlier osteotomy of the radius is to be seen. The *lateral deviation of the hand* persists due to abnormal projection of the ulnar head below the level of the shortened radius. Note divergence of the forearm bones. (B) Following subperiosteal resection, the lower end of the ulna has regenerated. The head is smaller and there is *medial deviation of the ulna*. Because of this, the axial parallelism of the forearm bones has been restored even though the difference in length has not been corrected.

correction of the underlying pathology. The other leads quite naturally to the simple expedient of resection of the projecting end of the ulna.[2] In one case in which this method was applied, a very interesting observation was made:

Case 1.—T. S., age nine, was brought to the hospital in November, 1932, because of a painless swelling of the left wrist. The mother stated that the child's wrist had been "twisted" some two months before. Examination disclosed prominence of the left ulnar styloid. The hand was held in volar and radial deviation. There was no pain or tenderness. No limitation of motion was observed in the hand or fingers. Measured from the tip of the olecranon to the radial styloid, the left forearm was about one and one-quarter inches shorter than the right. Roentgenograms showed a separation of the bones at the wrist. The radius was short. The distal radial epiphysis was wide, mushroomed, and sclerotic. The plane of the articular surface inclined acutely forward and medialward.

In the face of the marked bony deformity, it was apparent that the recent history of trauma was merely coincidental and not of etiologic significance. In fact, the patient was considered as suffering from a Madelung's deformity, and a manipulative correction of the deformity was unsuccessfully attempted. In December, 1936, because of the progressive radial deviation of the hand, osteotomy of the radius above the epiphyseal line was undertaken. Despite this, radial deviation of the hand persisted. Roentgenograms taken one year later, in December, 1937, (Fig. 1 A) disclosed a marked disproportion in the relative length of the forearm bones. The ulna projected not only below the level of the radial styloid but well beyond the proximal row of carpal bones, and even with the hand in radial deviation impinged against the carpal cuneiform. In October, 1938, a subperiosteal resection of the lower end of the ulna was performed, and the forearm was immobilized in plaster for a period of eight weeks. Thereafter, physiotherapy was instituted. The patient has noted complete return of power and has no limitation of motion. She notes occasional pain on change of weather. Examination discloses but slight prominence of the ulnar head on pronation. The wrist is wider than on the opposite side. Roentgenograms (Fig. 1 B), taken in December, 1939, disclosed the explanation of these phenomena. The lower end of the ulna has regenerated. Though its level with relation to the carpus has been changed but slightly, the ulnar head is definitely smaller and has clearly been deformed by medial pressure of the carpus. The fact that radial deviation of the hand disappeared after medial displacement of the ulna occurred seems to indicate clearly that it was the carpus and not the ulna which was "dislocated."

Though resection of the lower end of the ulna may indeed effectively eliminate the distressing prominence of its lower end, the possibility of injury to the ulnar collateral ligament with resultant disability is a danger to which Darrach has himself called attention.

From the functional point of view, the wrist is a compound joint, composed of the radiocarpal, the intercarpal, the meniscocarpal, and the radio-ulnar joints. In this complicated apparatus the head of the ulna forms a truly pivotal point. It is the point in relation to which the normal position of the other bony landmarks are determined and about which all of the motions of the wrist must be conceived of as occurring. It is the point to which are attached the ligamentous structures which fix the radiocarpal mass and thus insure free and forceful wrist motion. These ligaments comprise, roughly, three separate groups, which diverge fan-like from their ulnar origin. The first, the triangular fibrocartilage, is attached to the inferior surface of the ulnar head and serves to unite the ulna with the sigmoid notch at the lower end of the radius. The second, the ulnar collateral ligament, arises from the styloid tip, is firmly united to the base of the triangular fibrocartilage, and is inserted by two fasciculi into the cuneiform and pisiform bones. The third includes the anterior and posterior inferior radio-ulnar ligaments which arise from the lateral aspect of the ulnar head and bind it firmly into the ulnar notch on the radius. Loss of the integrity of either of these results in decreased fixation of the radiocarpal mass, with resulting prominence of the lower end of the ulna, and constitutes an indication for repair or reconstruction of the damaged ligaments, rather than ablation of the keystone upon which the functional integrity of the remaining structures depends.

The ulnar collateral ligament is probably the most important of these ligaments in stabilizing the wrist joint. Though the literature seems to indicate that injury to the triangular fibrocartilage is the primary cause of the weakness of the wrist joint,[9] it seems much more likely that this is frequently only coincidental to the more serious detachment of the ulnar collateral ligament.

The following case seen shortly after the reports of Mitchell,[8] and Gibson[4] is of interest in this connection:

Case 2.—J. J., age 17, was first seen in June, 1926, complaining of weakness and pain in the left wrist. The pain began on the ulnar side of the left wrist and radiated up into the forearm and down into the fingers. It was made worse by typewriting, so that the patient was forced to stop his work. He occasionally complained of weakness in the fingers and a clicking sensation on rotation of the forearm.

On questioning, it appeared that during a baseball game in 1924, the patient had suffered an injury while sliding into base with the outstretched left hand in full pronation. He complained of immediate pain and a marked prominence over the lower end of the left ulna. One of his friends "pulled his wrist out" and the patient felt something snap into place. Shortly thereafter he was seen by a physician, who stated that the patient had suffered a fracture of the wrist. Treatment consisted of splinting for a period of two months, after which the patient returned to work. In the early part of 1926 the patient began to notice pain and a gradual prominence of the lower end of the ulna.

Examination disclosed what appeared to be a marked hypermobility of the ulna, both forward and backward. On pronation, the ulna became definitely prominent on the dorsum of the wrist. As the hand was pronated and supinated, a soft click could be felt and heard over the head of the ulna. There was no limitation in motion of the wrist. On the right side, a radial deviation of the hand to 20° was possible. On the left side, this was possible to 35°. When the hand was examined in this position, there was a distinct depression to be felt just beneath the ulnar styloid. The whole hand could be abnormally displaced forward and to the radial side. There were no weakness or sensory disturbances in the hand or fingers. Clinically, there appeared to be no change in the position of the bony landmarks.

The roentgenograms were reported by Dr. A B. Ferguson as follows: "The styloid process of the ulna is separated from the shaft at its base. This condition is believed to be a developmental abnormality. No other variation from the normal is noted at this examination." However, it is to be noted that there is a definite anterior dislocation of the carpus and that the head of the ulna articulates with the radius at its normal level (Fig. 2 A and B).

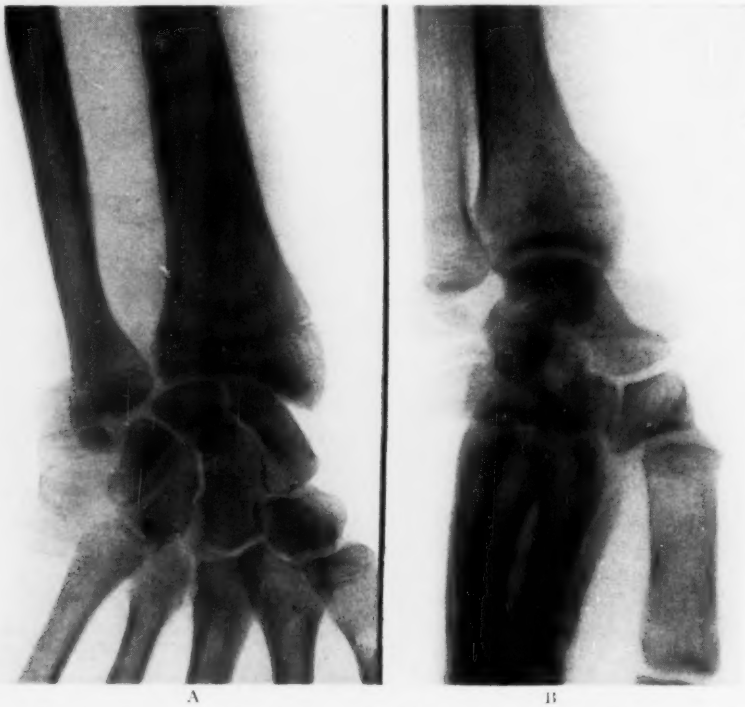
Despite this, it was felt that the patient had probably suffered a fracture of the ulnar styloid, with rupture of the ulnar collateral ligament and the attachment of the triangular fibrocartilage. Since the fracture of the styloid did not seem to be the cause of the symptoms, it appeared likely that the disability was to be attributed to the soft tissue injury and exploration of these structures was undertaken.

Operation.—July, 1936: Through a longitudinal ulnar incision, the course of the ulnar collateral ligament was exposed. The ligament was found attached to the tip of the styloid process. The triangular fibrocartilage, still partly attached to the ulnar styloid, was completely torn away from the inferior surface of the ulnar head. The relationship of the head to the radius appeared undisturbed. An effort was made to suture the fibrocartilage to the edge of the ulna, and a heavy chromic suture was taken through the styloid process to unite it and the attached ulnar ligament to the tip of the ulna. The wound was closed in the usual manner and a plaster of paris bandage

was applied. At the end of four weeks this was removed and gentle, active motion was begun.

The patient made an uneventful recovery and shortly after reported the return of normal use of the wrist.

Somewhat similar experiences have been reported in regard to isolated injuries to the inferior radio-ulnar ligaments. Disabilities due to disturb-



A B

FIG. 2.—Case 2: (A) The bones are of relatively normal length. There is a slight diastasis of the radio-ulnar joint but the ulnar head articulates normally with the notch on the radius. There is no radial deviation of the hand. (B) Lateral view discloses the dorsal prominence of the ulnar head, "so-called posterior dislocation of the ulna" simulated by anterior displacement of the radiocarpal mass.

ances in these structures present essentially the same symptoms and signs as are characteristic of the whole group of wrist joint derangements. In addition, however, the roentgenograms disclose a diastasis at the inferior radio-ulnar joint, the bones being of normal length. This, of course, is the pathognomonic sign, and the one which indicates the necessity for repair or reconstruction of the involved ligaments.

In 1926, the writer[6] devised a fascial loop operation designed to accomplish this purpose. The procedure seems to have met a definite indication, in the opinion of other surgeons. With but slight modification, it is almost identical with the operations later described both by Eliason,[3] and by Lowman.[5] However, attention must be called to the important fact that this technic is indicated, when and only when, the luxation occurs without loss of the normal bony alignment. It is intended strictly for repair of the

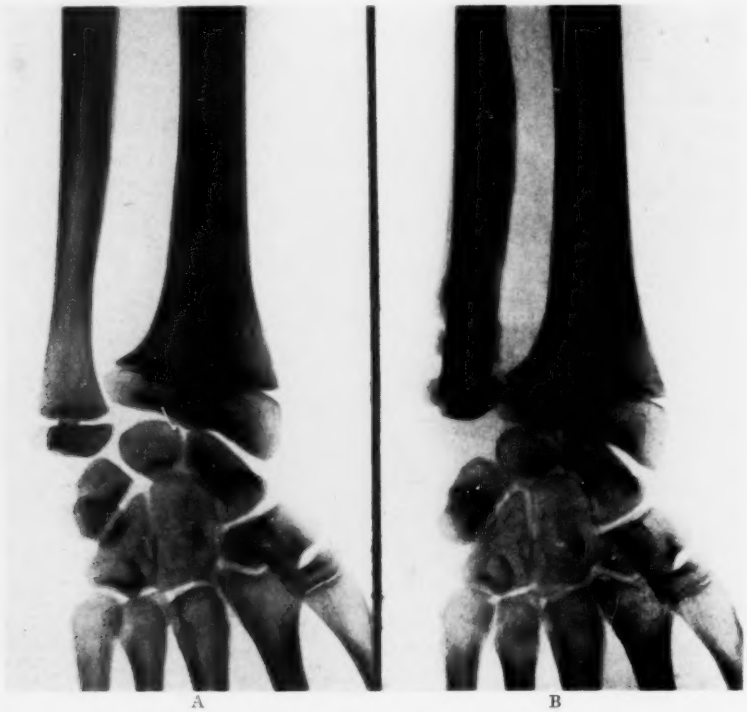
ligamentous apparatus and can have no successful application unless the bony disproportion has been previously corrected. Indeed, the necessity for reestablishing normal bone relationships before undertaking any ligament reconstruction, formed the basis of the conversation to which Lowman referred in his report of 1930.

The bone disproportions which may be found in derangement of the wrist fall into three main groups. Each presents the general features of the larger class of wrist derangements and, in addition, at least one distinguishing sign, which justifies its special consideration. The first of these groups is characterized by a loss of parallelism, an angular deviation in the axis of one or the other of the two bones of the forearm. Most commonly the radius is the site of angulation such as is found in Madelung's deformity, or in a malunited fracture. Occasionally, however, the deformity may be caused by disease or malunion in the ulna. As regards the inferior radio-ulnar joint, the effects from the involvement of either of these bones may be identical and the only difference will be in the point of application of the corrective forces. In this type of case, simple osteotomy, for correction of the malalignment, is usually, but not invariably, sufficient to overcome the disability, as Campbell,[1] and others have pointed out. In those cases in which additional repair of the ligaments is necessary, the osteotomy constitutes an essential preliminary step.

The second group is characterized primarily by a disproportion in the relative lengths of the two forearm bones. Normally, the ulnar notch on the radius is approximately at the same level as the ulnar head, while the radial styloid projects at least one-half inch beyond the ulnar styloid. As a consequence of the greater length of the radius, the lower end of the ulna is at some distance from the carpus, so that any interference with pronation and supination is avoided. However, when this arrangement is disturbed and the ulna projects beyond the level of the radius, the ulnar head impinges against the carpus, with resultant limitation of rotation and subsequent relaxation of the ligamentous fixation of the wrist. This may occur in other conditions (see Case 1), but it is typically an end-result of improperly reduced Colles fractures and seems to be the cause of the disability to which so much attention has recently been directed.

With the object of determining this point, a number of cases of Colles fracture were examined. In those in which symptoms of weakness persisted, it was found that a relative shortening of the radius was an almost invariable finding. Careful examination of the radiographs in such cases demonstrated that the ulnar head projected well down over the shadow cast by the carpal bones. The impression was gained that in rotation of the wrist, the carpal bones impinged against the projecting ulnar head and forcibly caused its dislocation. It seemed reasonable to believe that the cause of the so-called luxation lay primarily in the bone block, and that elimination of the osseous disparity would lead to disappearance of the symptoms.

The opportunity for testing the validity of this conception was offered by several patients, who presented themselves for the treatment of post-fracture "ulnar dislocations." Though specifically admitted with the intention of performing a fascial graft reconstruction, roentgenographic study suggested the desirability of preliminary shortening of the ulna by a cuff resection[7]. The following typical case proved that no ligamentous reconstruction operation was necessary:



A B

FIG. 3.—Case 3: (A) The hand is displaced radialward. The ulnar head projects below the level of the radial notch and clearly impinges on the carpal cuneiform. (B) Union has occurred after subperiosteal cuff-resection through epiphyseal line, with shortening of the ulna. The head articulates with the radial notch and no longer impinges against the carpus.

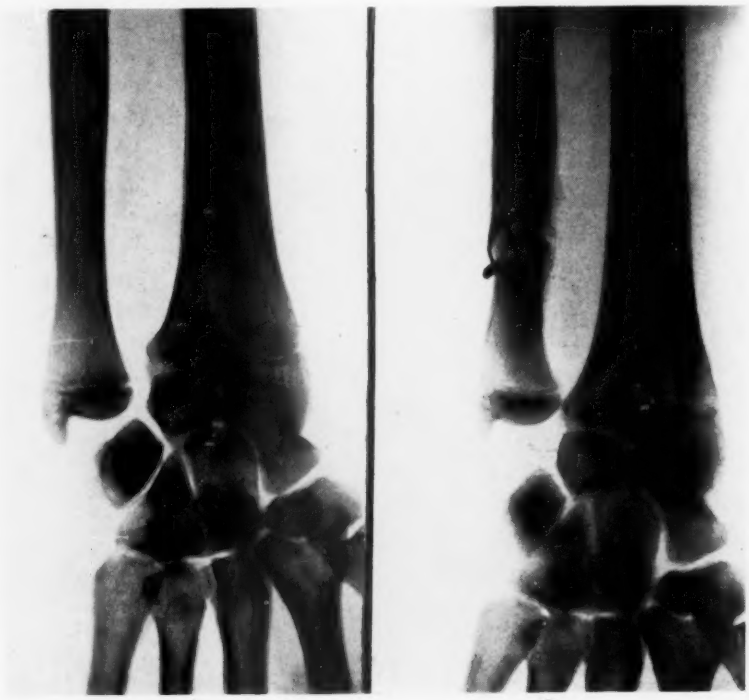
Case 3.—T. H., age 16, appeared in the Out-Patient Department, in April, 1939, complaining of weakness in grasp and difficulty in rotating his left forearm. In January, 1926, while sleigh riding, the patient had suffered a fracture of the left wrist, but no attempt at reduction had been made. The patient noted no trouble with his wrist until several months ago, when he began to observe a decreasing power of grasp and prominence of the lower end of the ulna.

Examination disclosed a radial deviation of the hand. The lower end of the ulna was prominent and at a level below the normal in relation to the radial styloid. Extension at the wrist was normal. Flexion was limited at 55°. Supination was about three-fourths normal. On pronation of the hand, the prominence of the lower end of the ulna became markedly exaggerated. Both ulnae measured 9¼ in. The right radius measured 9¼ in.; the left only 8¾ in.

Roentgenograms, taken May 8, 1939, (Fig. 3 A) showed "an old oblique fracture of the anterior aspect of the distal end of the left radius, just proximal to the epiphyseal

plate. There is a slight separation of the distal end of the left radius, just proximal to the epiphyseal plate. There is a slight separation of the distal epiphysis of the ulna and a separation of the distal radio-ulnar joint. There is a definite disproportion in growth between the ulna and the radius, with the ulna projecting below the radius. The epiphyseal lines are not closed."

Operation.—May 22, 1939: Under general anesthesia, a two-inch incision was made over the lower end of the left ulna. The bone above the epiphyseal line was exposed subperiosteally. A block of bone, including the epiphyseal plate, measuring about three-



A B

FIG. 4.—(A) Case 4, previously reported.[7] As a result of fracture, the articular plane of the radius is abnormally inclined medially and the ulnar head projects below its normal level. (B) After subperiosteal cuff-resection, above the epiphyseal line, the ulna has been shortened so that its head articulates with the radial notch. Despite the abnormal medial angulation of the articular surface, normal function of the wrist was restored.

quarters of an inch, was resected. Holes were drilled in distal and proximal fragments, which were then drawn together, with chromic sutures. The periosteum was reunited, and then the skin wound was closed. A plaster of paris bandage was applied from the fingers to the midarm, with the elbow fixed at right angles and the forearm in mid-pronation.

On June 22, 1939, the plaster encasement was removed. Roentgenograms taken at this time (Fig. 3 B) showed union of the fragments. The longitudinal alignment was good, but there was a moderate rotation of the ulnar fragment. The patient was given physiotherapy. In October, 1939, it was noted that the correction was excellent. There was no evidence of "so-called" dislocation of the distal end of the ulna and no weakness, despite the rotation of the distal ulnar fragment.

Case 4.—This case has been previously reported[7] (Fig. 4).

In Case 3, the cuff-resection was purposely planned so as to include the epiphyseal plate. Except where it is intended to prevent further growth

of the ulna, it is better to perform the operation about one inch or one and one-half inches above the head of the ulna. This facilitates the fixation of the lower fragments and precludes the possibility of interference with radio-ulnar motion by excess callus formation, at the joint level. The plane of the resection may be right angles to the shaft (Fig. 4 A and B) or may be made oblique.

Experience in the treatment of Colles fracture has shown that the disproportion in the lengths of the forearm bones which is due to impaction and relative shortening of the radius is of the most serious consequence. The limitation of motion which is occasioned by improper alignment of the inferior radial surface is of comparatively slight importance. While every effort should be made to correct all deformities during the initial setting of the fracture, far too much attention is devoted to reposition of the articular surface and far too little to restoration of the relative length of the radius. Where this is overlooked, "dislocation" of the ulna, pain, weakness, and limitation of rotation result from the impinging of the ulnar head against the carpal cuneiform.

In young children the disproportion in length between radius and ulna may be prevented or partly overcome by fusion of the lower ulnar epiphysis so as to arrest its growth. However, in adolescents near the age of normal epiphyseal closure, and in adults in whom the epiphyseal line has already been obliterated, the growth arrest operation is, of course, not applicable. In such instances simple shortening of the ulna to restore the anatomic proportion between the bones is sufficient to obviate the symptoms and usually precludes the necessity of any further operative reconstruction of radio-ulnar ligaments.

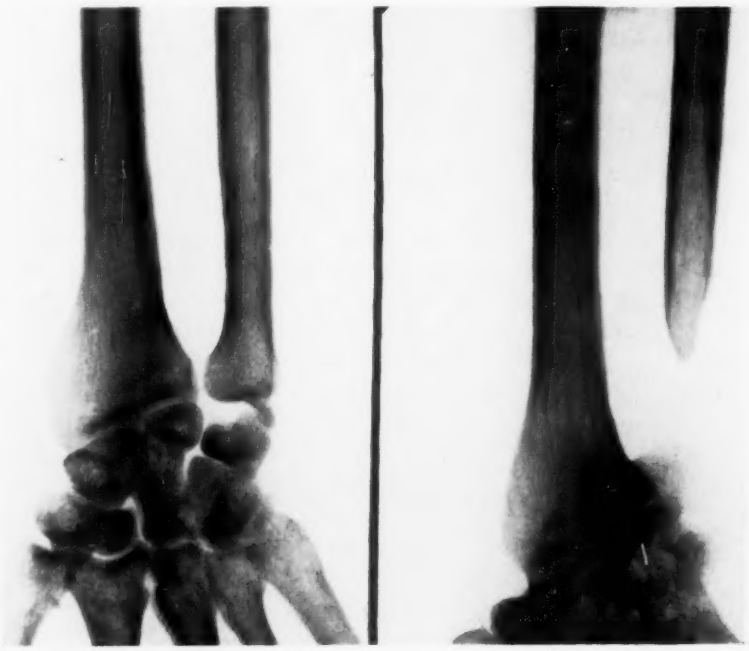
The third group is that in which the ulnar prominence is due either to arthritis at the radio-ulnar joint, or to disease of the ulnar head. In this event, of course, the hope of retaining the function of the radio-ulnar joint is illusory and resection offers the most expeditious form of therapy. Under certain circumstances, however, it may be desirable to preserve the ulnar head and its attached ligaments. In such cases, fusion at the radio-ulnar joint, with the subsequent formation of a pseudo-arthrosis by cuff-resection of the ulna, just above the head, is suggested. This principle of treatment was employed with success in the following case:

Case 5.—M. W., female, colored, age 28, entered the Out-Patient Department, December 31, 1934, complaining of a painful swelling of the right wrist of four days' duration. Gonococcal infection was denied. There was a history of influenza two weeks before the onset of symptoms, and it seemed reasonable to believe this was the origin of the infectious arthritis of her wrist. Nevertheless, in the course of routine investigation, the patient was found to be suffering from a bacteriologically established endocervicitis. A moulded plaster of paris cock-up splint was applied to the hand and forearm, with much relief to the patient.

In May, 1935, it was noted that ankylosis of the right wrist had occurred, with the hand in 15° of flexion. Rotation of the forearm was markedly limited. Supination was possible to only 15° beyond the neutral position. The lower end of the ulna projected

posteriorly and appeared to be subluxated. Roentgenograms showed absorption of the articular cartilage covering the carpal bones, the wrist, and the radio-ulnar joints (Fig. 5 A). To correct the palmar flexion and the pronation deformity, open operation was decided upon.

Operation.—December 19, 1935: Through a dorsal incision, the wrist joints and the lower end of the ulna were exposed. The ulnar head was dislocated posteriorly and the radio-ulnar joint was filled with connective tissue. The radiocarpal joint was the site of a destructive arthritis. The radio-ulnar joint was cleaned out and the dislocation of the ulnar head was overcome. The lower portion of the ulna was subperiosteally exposed and one and one-half inches of the bone above the head was



A B

FIG. 5.—Case 5: (A) Arthritis of the wrist with dorsal prominence of the ulnar head. The carpus is elevated and impinges against the ulna. The relative length and axial parallelism of the bones is retained, with barely any radio-ulnar diastasis. (B) Postoperative. The radiocarpal and radio-ulnar joints have been fused. To restore the power of rotation, a cuff-resection of the ulna has been performed.

resected. The ends of the bone were covered with a muscle flap. The wrist joint was then opened and thoroughly curetted. The wrist was brought into dorsal extension and the position was maintained by sutures taken through the adjacent bone surfaces. The wound was closed without drainage, and a plaster of paris bandage was applied, with the forearm in supination and the wrist in 25° dorsal extension. The postoperative reaction was uneventful and the patient was discharged one week after operation.

The usual ambulatory treatment was carried on in the Out-Patient Department. As early as May, 1936, it was noted that the patient had solid fusion of the wrist, with return of excellent pronation and supination. The prominence of the ulnar head had completely disappeared. Roentgenograms, in December, 1936, revealed the fusion and the area of ulna resected (Fig. 5 B). Examination, in April, 1939, disclosed the persistence of painless rotation. The only complaints were directed to pain and stiffness in the fingers, which were involved in the arthritic process.

CONCLUSIONS

So-called dislocation of the lower end of the ulna is a misnomer. Prominence of the ulnar head is a characteristic of a number of different conditions, which are more accurately to be designated as derangements of the wrist joint.

The head of the ulna may be considered as a fixed point about which the motions of the wrist joint occur. To it are attached the ligamentous structures which stabilize the wrist and which are necessary to its normal function.

Prominence of the ulnar head may be caused by:

1. Injury to the triangular fibrocartilage.
2. Injury to the ulnar collateral ligament.
3. Rupture of the radio-ulnar ligaments.
4. Axial deviation of either of the forearm bones.
5. Disproportion in length of the forearm bones.
6. Enlargement or disease of the ulnar head.

Except for specific indications, the ulnar head should be spared and surgical attention should be directed toward repair or reconstruction of the ligamentous apparatus.

Repair of the soft tissues of the wrist can only be undertaken successfully if the bony relationships are normal, or have been previously restored to normal by appropriate types of osteotomy. For axial malalignment, simple linear osteotomy of the involved bone is sufficient. For disproportion in length, shortening of the ulna has proven extremely satisfactory.

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THE TREATMENT OF FRACTURES OF THE OLECRANON BY LONGITUDINAL SCREW OR NAIL FIXATION

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The indication for operative reduction and internal fixation in fractures of the olecranon with separation of a sizable fragment is generally recognized. It is agreed, too, that open surgery with fixation of the fragments is required in comminuted fractures of the olecranon with displacement, and in fractures complicated by anterior displacement of both bones of the forearm. Until within the past few years the writer has been accustomed to use sutures of absorbable material as a means of fixation. A method that is proving to have certain advantages over such sutures is the fixation of the fragments by a longitudinal screw or nail. Not only is it possible to obtain reduction with greater accuracy and to ensure better retention of the fragments by this form of anchorage, but the period of disability is materially shortened. To the working man this decrease in the time element in recovery is of particular value.

Effective treatment of fractures of the olecranon depends upon two factors: (1) Accurate anatomic reposition of the fragments; and (2) sufficient fixation to permit immobilizing the elbow at a right angle. If the fragments are not replaced accurately, so that bony union takes place with perfect apposition, the power of complete extension may be lost. Moreover, as the fracture penetrates into the joint, any roughness or irregularity of the articular surface is not well tolerated, and arthritic changes will eventually develop. Following the reduction and fixation of the fragments, the elbow must be immobilized at a right angle, the position that is favorable both to healing and to the recovery of motion. The practice of splinting fractures of the olecranon in the position of extension, which still exists, is poor fracture therapy. In such cases, the convalescence is prolonged and the power of flexion is regained slowly. It may happen that, under this management, the fragments will separate upon the attempt to flex the elbow, or that complete flexion will never be recovered because of contracted tissues.

In the fulfillment of the requirements of efficient treatment of fractures of the olecranon, the use of a screw or nail for fixation is an advance over absorbable sutures. In a transverse or oblique fracture it is possible to obtain a hair-line apposition of the fragments and to fix them more tightly than when sutures are used. Particularly is this true when a screw is introduced, for, as it is threaded into position it tightly engages the fragments and closes the fracture-gap. In comminuted fractures, the main olecranon fragment can first be aligned with the ulnar shaft, and then the smaller fragments fall into position easily. In fractures of the olecranon associated

with anterior displacement of the forearm bones, in which accurate reduction and retention of the fragments are essential to the control of the reduced dislocation, the longitudinal fixation ensures perfect reposition.

Once this fixation material is in place, there need be no hesitation on the part of the surgeon in flexing the forearm to a right angle. In contrast, when absorbable sutures are used, there is the danger that they will not

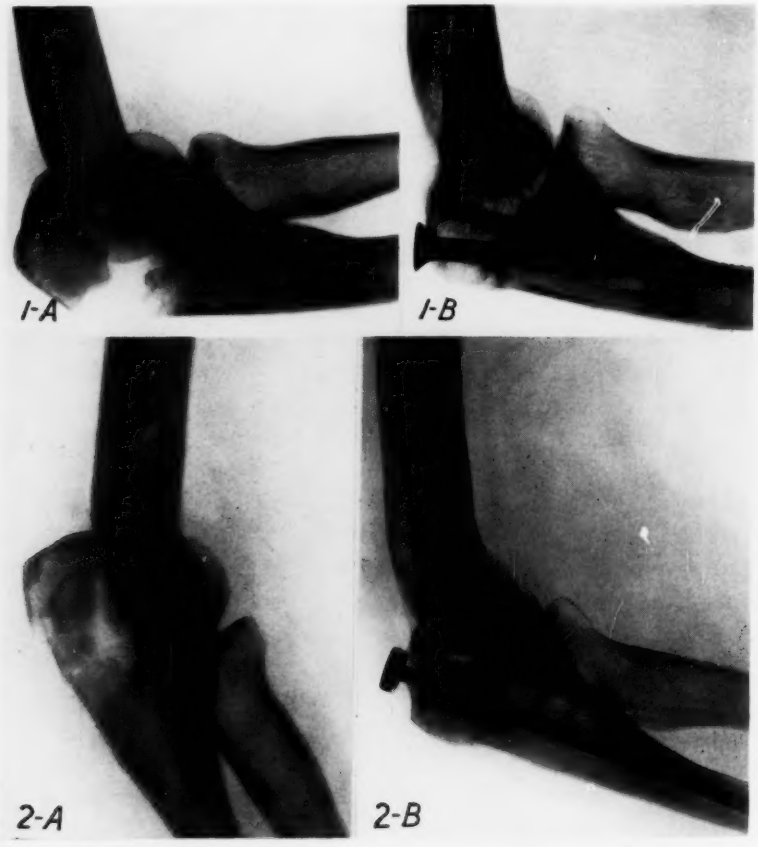


FIG. 1.—Patient H. S.: (A) A transverse fracture of the olecranon with separation of the fragments. (B) Postoperative roentgenogram showing fixation of the fragment with a longitudinal screw.

FIG. 2.—Patient L. D.: (A) A comminuted fracture of the olecranon with displacement. (B) Roentgenogram taken five months after reduction, with longitudinal nail in place.

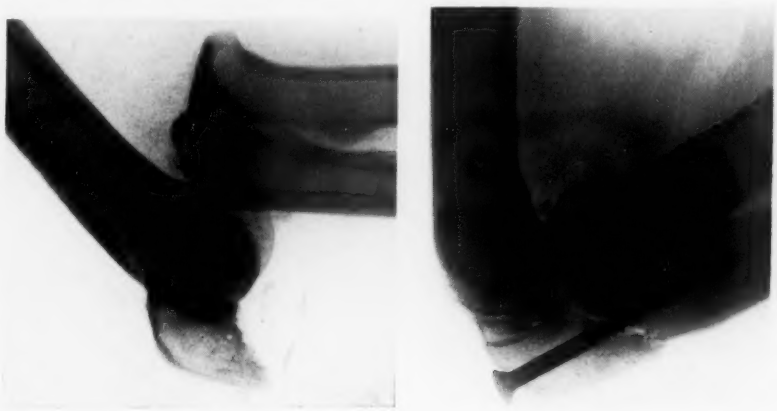
withstand the tension and that the fragments will separate as the forearm is flexed to the right-angle position for immobilization.

The great advantage of internal fixation by this means is the shortening of the convalescence. So accurate is the replacement of the fragments and so secure is their retention in position that motion may be started in from four to five days after the reduction. This is in contrast with the three or four weeks of immobilization that is necessary following fixation by other methods.

The prompt healing affords relief from pain and soreness, so that the patient has the confidence to start motion early. Within a few weeks a good arc of motion is possible, whereas, in the case of sutured fragments the convalescent period extends over several months.

Operative Technic.—The operation is best carried out in from three to five days after the injury, when the swelling will have subsided. A general anesthetic is administered and a tourniquet carefully applied.

A longitudinal incision is made, beginning one inch above the tip of the olecranon and extending downward to a point from one to one and one-half inches below the fracture-cavity. The incision is carried down to the periosteum, and the skin and subcutaneous tissues are retracted laterally to expose the fracture-cavity. The blood clots, tabs of torn periosteum, and minute detached spicules of bone are removed. Any fragment that retains sufficient attachment to ensure a blood supply is not disturbed. The ends



A B

FIG. 3.—Patient R. J.: (A) A fracture of the olecranon with anterior displacement of both forearm bones. (B) Postoperative roentgenogram showing fixation of the fragments with a longitudinal screw.

of the fragments are cleaned with pledgets of gauze. The forearm is extended, and the replacement of the fragments studied.

The forearm is then flexed about 15 degrees, and a short longitudinal incision is made in the triceps tendon above the tip of the olecranon. A small drill is inserted in the proximal fragment, at the tip of the olecranon, and forced through the fragment into the center of the fracture-cavity. The fragments are approximated and held in position by means of a tenaculum.

A screw of sufficient length to fix the fragment to the ulnar shaft is passed through the drill-hole and threaded into the ulna. If possible, it is well to so direct the screw that it engages the distal cortex of the ulna, thus providing for perfect fixation (Fig. 1 B). If a nail is used, it must be sufficiently long so that its tip engages the cortical bone beyond the bow of

the ulna (Fig. 2 B). The lateral expansions of the triceps tendon are sutured with interrupted chromic catgut.

In comminuted fractures, when the main fragments have been aligned, the smaller ones are easily fitted into place. In the case of fractures of the olecranon associated with displacement of both bones of the forearm, the dislocation is first reduced and then internal fixation of the ulnar fragments is established.

Following the closure of the wound, the elbow is immobilized at a right angle. A posterior plaster shell may be applied and worn for a few days, but it is sufficient to use a tight bandage and carry the arm in a sling. Exercises of the fingers and the shoulder are started on the second day, the patient squeezing a rubber ball and abducting the arm hourly. Through such exercises the tonicity of the muscles of the arm and forearm are preserved. Gentle motion of the elbow, in flexion and extension, can usually be started on the fourth or fifth day after reduction. At this time hot fomentations may be of help.

The insertion of the screw or nail is a simple procedure and is easier, in fact, than the fixation of the fragments with sutures. The screw or nail may be removed in about six months, but this is optional.

CONCLUSIONS

Fixation of the fragments in olecranon fractures by a longitudinal screw or nail is a simple procedure that has certain advantages over the suture methods in general use. By this method it is possible to obtain reduction with greater accuracy and to ensure better retention of the fragments. Good functional recovery is established within the shortest possible time when this longitudinal fixation is used, a factor of importance to the active adult.

THE REGENERATION OF JOINT TRANSPLANTS AND INTRACAPSULAR FRAGMENTS*

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IN DISCUSSING joint transplantation, it is important to distinguish between transplantation of entire joints and transplantation of one-half of a joint; furthermore, between autogenous and homologous transplantation. While transplantation of entire joints was soon given up as impractical, transplantation of one epiphysis or one-half of a joint, particularly if done autogenously, became a well-established procedure.

Tuffier[31] (1901) was, supposedly, the first surgeon who performed a joint transplantation. In a fracture through the surgical neck of the humerus, he removed, temporarily, the head of the humerus and reimplanted it on the humeral-shaft in a more favorable position. Tietze,[30] however, as far as can be ascertained, was the first who performed a genuine joint transplantation; he replaced the resected lower part of the radius with a phalanx of a great toe. The first experimental studies upon this subject were performed by Judet,[16] in 1906. He studied the condition of the transplanted cartilage in rabbits and found the cartilage preserved in those cases in which he performed a reimplantation, *i.e.*, an autogenous transplantation of joint surfaces. In a second series of experiments he performed homologous transplantations of entire or half-joints, with or without capsule. The joints were transplanted beneath the skin of the other animal. In cases where the naked joint was transplanted, erosion of the graft by fibrous tissue occurred while the grafts transplanted with the capsule remained preserved. Impallomeni,[15] Ducuing,[10] and Voronoff[32] had similar results. Dalla Vedova,[9] however, observed a complete necrosis in those parts which were transplanted with the joint capsule, but large parts of those grafts transplanted without capsule remained alive.

It must be assumed that the most favorable conditions in joint transplantation will be those in which only one-half of the joint is replaced and the capsule of the host joint remains preserved. Gill[12] and Haas[13] proved this in autogenous joint transplantations, and Rehn and Wakabayashi[24] in homologous transplantations. Gill autotransplanted the second long metatarsal bone in dogs from one paw into the opposite paw. After seven to eight months he found the articular surfaces normal and the joints had perfect function; the bone showed no evidence of necrosis. Haas, in a large series of experiments, studied the effect of transplantation of the epiphysis on bone and cartilage regeneration and the bone growth after reimplantation and autotransplantation of metacarpal and metatarsal bones in dogs. He concluded that the articular cartilage offers the greatest possibilities for successful transplantation of the various parts of a bone. The bone itself degenerates but later becomes regenerated from periosteum and endosteum, as Phemister,[22] and others, have

* Read before the Philadelphia Orthopedic Club, January 9, 1941, at Philadelphia, Pa

described it. The longitudinal growth stopped in every case because the epiphyseal line ceased to function. V. Tappeiner[29] came to a similar conclusion. Rehn and Wakabayashi—even in homologous transplantations—found the epiphyseal cartilage fully maintained in function and histologic structure after the transplantation.

After having studied the results of animal experimentation, it is interesting to compare them with joint transplantation in human beings. Lexer[19] (1907) was the first who performed transplantations of entire joints together with menisci and crucial ligaments—however without capsule. This startling operation, mostly performed for replacement of ankylosed joints, was soon given up by its originator and replaced by simpler operations; namely, the joint plastic with interposition of fat tissue or fascia. Nevertheless, of the 23 cases in which Lexer performed such homologous transplantation, 12 cases resulted in the transplant healing and mobility remained for a number of years. Later, however, mobility decreased and became painful due to extensive arthritic changes. Two of those patients in whom entire knee joints had been transplanted—the transplant for one was taken from an executed criminal—could be followed for 14 and 16 years, respectively. Bürkle-de la Camp[6] later examined the specimens. The joint cleft was still preserved, but the cartilage was replaced by fibrous tissue, and there was extensive subarticular break-down of the subchondral cancellous bone. These consequences are apparently less serious in homologous half-joint transplantations where only one-half of the joint is replaced and the host capsule remains preserved (Lexer, Enderlen[11]). They are, as shown by a number of authors, entirely absent in autogenous half-joint transplantations provided proper operative and postoperative precautions have been taken. Rovsing[25] (1910) successfully replaced the upper two-thirds of the humerus with the upper part of the fibula in the same patient. Klapp[17] (1912), after a similar operation, followed his patient for seven and one-half years; there was hardly any limitation of function. Albee[1] reports several cases in which he resected the upper humerus for primary malignant tumors and grafted the upper part of the fibula into the defect. One patient was followed for nearly 17 years; there was good function and no evidence of recurrence. A number of similar cases have been published since, replacement of the upper half of the femur and the lower half of the radius by the upper half of the fibula; replacement of phalanges and metacarpal bones by bones of the foot (Behrend,[3] Phemister,[22] Portugolow,[23] Skillern,[27] *etc.*). But only rarely have specimens of these autogenous grafts been obtained for investigation of the regenerative processes. Conclusions gained from animal experiments are of doubtful value in those cases in which graft and host bone cannot be immobilized. More reliable results come from those experiments in which entire bones are transplanted (Gill, Haas).

I[20] reported recently about an experiment where I took out entire radii in dogs and returned them into their original places. Thus a transplantation of bones was combined with a transplantation of epiphyses. After injection

of the bone vessels with contrast material and visualization of the vessels roentgenographically, the specimens were examined microscopically. This examination showed that the bony parts and the medullary tissue died after transplantation. Later on, however, from two and one-half to four months postoperatively, the dead graft was transformed gradually into living bone tissue by osteoblasts which accompanied the periosteal vessels on their way through the haversian canals of the graft. Of the transplanted cartilages, the outer layer remained alive. Considerable parts of the deeper layers, however, were found dead primarily, but later stages showed these layers perfectly regenerated.

I should like to present a clinicobiologic study of the regeneration of joint transplants, and of certain intracapsular fragments, and shall attempt to parallel the two.

REGENERATION OF JOINT TRANSPLANTS: CASE REPORTS

Case 1.—L., age 43 (referred by Dr. Charles F. Mitchell), was admitted to the Germantown Hospital in January, 1940, with a fracture-dislocation of the head of the right humerus (Fig. 1). After several unsuccessful attempts of closed reduction, an extension dressing was applied to the right arm; an open reduction had been planned, but was postponed because of an extensive thrombophlebitis of the veins of the volar surface of the right arm. The operation was carried out March, 1940. The shoulder joint was opened by an incision that ran along the median border of the deltoid muscle and separated the clavicular insertion of this muscle (Henry, Thompson). The entire median half of the deltoid muscle was reflected laterally and backward, establishing an excellent exposure of the shoulder joint region. The next step was exposure of the head fragment. The head of the humerus could be felt beneath the pectoralis minor muscle. With considerable difficulty it was dissected free and temporarily removed. It was kept in normal salt solution for the time being. Now followed the subperiosteal exposure of the shaft of the humerus. After its stump was freed from cicatricial tissue, the head of the humerus was placed upon the shaft. A canal was drilled through shaft and head, the long biceps tendon was divided, threaded through it and sutured, according to Nicola. Thus the head of the humerus was not only fastened to the shaft of the humerus, but, also, the entire humerus kept in the glenoid. This fixation, however, was not sufficient. Therefore, I drilled two Kirschner wires through head and shaft in different directions, thus achieving an internal fixation of this fracture. The wound was closed in layers, the arm immobilized in right-angle abduction by a plaster encasement which included chest and the entire arm. The wound healed primarily, and the patient was discharged four weeks after the operation.

Five months after the operation the encasement was removed. Roentgenograms showed definite signs of union. Physiotherapy and motion exercise was started. About four weeks later the patient was readmitted. He stated that he had experienced a "fainting spell" (of alcoholic nature), and had fallen and broken the right arm again. This time the fracture was between the proximal and middle third of the shaft, while the former fracture remained firm. Judging from the size of the accompanying hematoma the fall must have been violent. The fragments were considerably displaced in angulation. This fracture was treated in extension with a Kirschner wire through the olecranon. It healed in good position (Fig. 1 b and c).

While the patient was in the hospital he developed a small superficial ulcer in his former scar. A prominence could be felt beneath this ulcer which was thought to be due to one of the wires working its way out of the replanted humeral head. For this reason operative removal was advised. I performed this operation in October, 1940

(seven months after the first operation). The shoulder joint was exposed through the lower part of the former incision. No evidence of a true capsule could be detected. The head of the humerus was firmly united with the humerus. The head of the long biceps tendon was found incorporated in the bone. There was no evidence of slipping of either wire. Although their upper canals were visible it would have been difficult to remove them; and for this reason they were left in place. The prominence was due to callus formation; it was levered off. Two small pieces of the replanted head of the humerus were taken for microscopic examination. One consisted of cartilage together with a thin

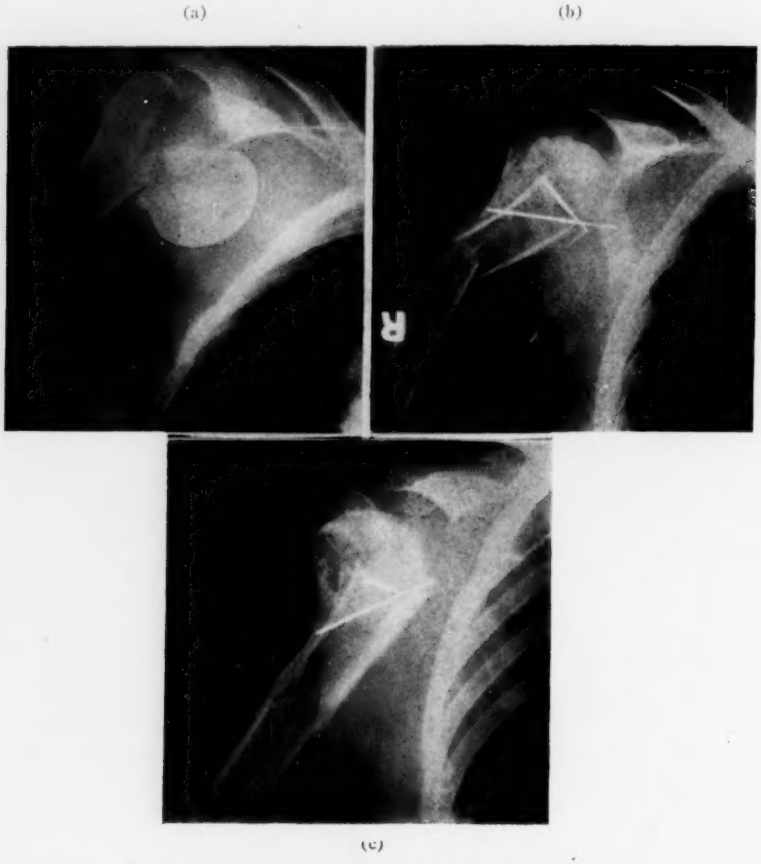


FIG. 1a.—Fracture-dislocation of head of right humerus.
b. Six months after temporary removal and reimplantation of head of humerus. Fracture of shaft of humerus after recent trauma.
c. One year and two months after the fracture-dislocation.

layer of underlying bone; the other one was taken from the same place but from deeper inside. Profuse oozing was encountered from both cut surfaces proving that the circulation of the head of the humerus had become reestablished. The patient was soon discharged to receive ambulatory physiotherapy. He has regained 50 per cent of the motility of his right shoulder in all directions, and is free from pain. He is employed as a truck driver and has recently lifted a trunk, weighing 155 pounds, from the truck to the scale and back again.* The patient was reexamined recently (two years after the operation). Roentgenologically the head of the humerus shows some "mushroom" deformity. Clinically and symptomatically there was no change.

* The histologic examination of the two specimens taken from the replanted head, seven months after the operation, revealed the following picture: A number of the lacunae of the bone were found empty, but in many other places the lacunae were filled with a well stained nucleus. The haversian canals contained blood vessels which were accompanied by osteoblasts. Rows of osteoblasts were also found along the trabeculae. On these trabeculae the difference between the living, *i.e.*, the newly formed bone, and the dead bone was clearly visible and there was no evidence of osteoclastic bone destruction and absorption. The medullary spaces were filled partly with a vascular fibrous tissue, partly with normal medullary tissue; beneath the cartilage even myeloid tissue could be seen. The cartilage itself was found alive in its outer layer while the middle and deep layer showed empty lacunae in various regions.

COMMENT.—An operation was performed for a fracture dislocation of the head of the humerus; during this operation the head of the humerus was temporarily removed and then replanted on the humerus shaft. Thus the head of the humerus could be compared with a half-joint transplant. It must be assumed that due to the complete interruption of circulation, the graft had died. But after its reimplantation vessels must have grown into the dead graft. The vessels were apparently derived from the shaft of the humerus and accompanied by intramedullary osteoblasts. These osteoblasts transformed the dead graft into living tissue by the so-called creeping substitution of the dead bone by new bone, as biopsy specimens, removed from the head of the humerus seven months after its reimplantation, revealed. The microscopic picture of these specimens were very similar to those which I[20] described in my former experimental work. The joint cartilage remained alive in its outer layer while sections of the deeper layers died. Thus, this autogenous joint transplant died in most of its parts, but was gradually regenerating from ingrowth of vessels and osteoblasts of the living bone; the ingrowth of these vessels was facilitated by a prolonged complete immobilization of the fragments.

This case is a typical example of an autogenous half-joint transplantation. Half-joint transplantation has become a well-established procedure. The autogenous transplantation within the same individual is preferred to the homologous transplantation due to better and quicker healing conditions. The disadvantage of the autogenous transplantation, however, is the limitation of the material to be transplanted; namely, the phalanges of the toes, the metatarsi and the fibula. So, for instance, the humeral part of the shoulder joint and the radial part of the wrist joint can successfully be replaced by the upper part of the fibula. Even the upper half of the femur has been successfully replaced by the fibula. If it comes to broader joint surfaces, however, particularly those which carry a socket, an autogenous transplantation cannot be performed. In those cases—if a half joint transplantation is considered at all, and a suitable donor available—the homologous transplantation is the only possibility. The following example, a short preliminary report which appeared in Brun's Beitr. z. klin. Chir., **160**, 30, 1934, might be recorded.

Case 2.—Female, age 23, was admitted to the hospital, complaining of throbbing pains in the right knee joint of six months' duration. Examination revealed a tumor

within the lateral half of the upper fourth of the right tibia (Fig. 2 a). The tumor had apparently broken through the cortex. There was no evidence of metastases. An amputation of the right lower extremity above the knee joint was planned in case the biopsy revealed a malignant tumor. The amputation, however, was refused by the patient. At the same time a male, age 70, was in the hospital who suffered from an arteriosclerotic gangrene of his right foot, with partial blocking of the popliteal artery, necessitating an amputation above the right knee joint.

A compromise operation was now planned for the first patient. Permission for such an operation was given. Both patients had blood Group O. The operation was carried

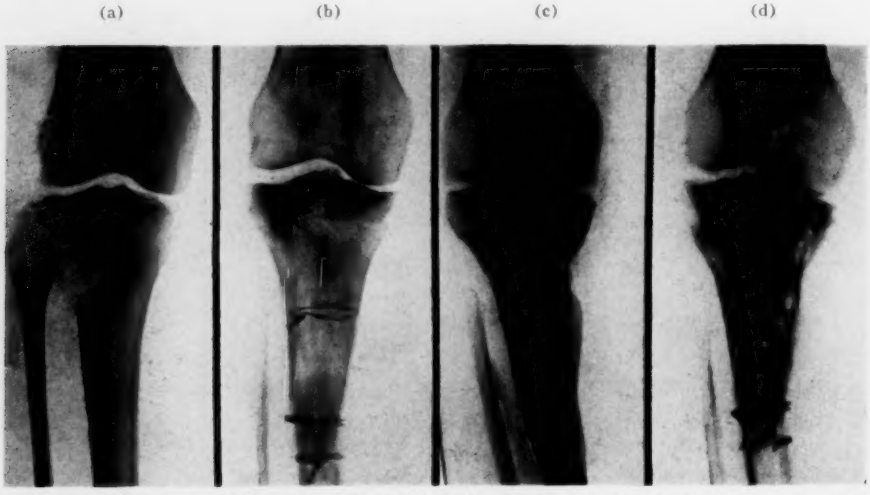
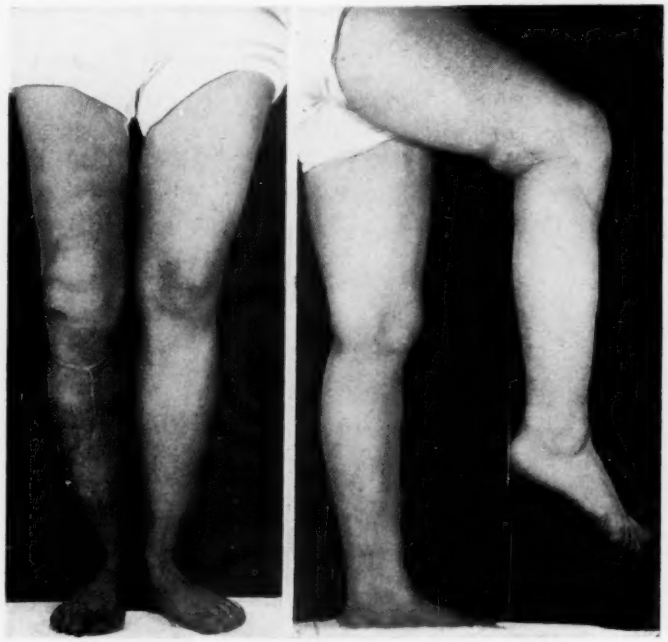


FIG. 2(a).—Sarcoma of lateral condyle of right tibia with invasion of soft tissues in a 23-year-old female.
(b). Same patient eight months after resection of upper third of tibia and transplantation of the corresponding part of tibia from a 70-year-old man. Head of fibula and surrounding soft tissues had been removed at the same time.
(c). Same patient two years after operation. Fusion between tibia and graft.
(d). Same patient five and one-half years after operation; no evidence of recurrence; subarticular breakdown of median condyle of graft. Roughening of articular surface.

out in June, 1933 (operators, Lexer-May). The biopsy confirmed the diagnosis of sarcoma. From a Y-shaped incision in front of and below the right knee (Fig. 3 a), the joint was opened after separation of the tibial tubercle, and lifting of it upward together with the skin flap and quadriceps tendon. The tumor was now exposed. It had penetrated into the soft tissues near the head of the fibula. Then followed the separation of the crucial ligaments and of the median and lateral ligaments at their insertion at the tibia. The head of the fibula with surrounding soft tissues, including one branch of the peroneal nerve and the upper third of the tibia, were now resected. The tibial resection was staggering. At the same time, in the same operating room, the corresponding part of the tibia of the male, age 70, had been resected in a similar way and was now transplanted into the girl's leg and fastened there with two wire loops. The lateral and median ligaments were attached to the tibia transplant as well as possible. The quadriceps tendon together with the tubercle of the tibia were fastened to the transplant with a wire suture. The wound was closed in layers and the leg placed in a plaster encasement reaching from the toes to midthigh. Healing was uneventful. Eight weeks after the operation the plaster encasement was replaced by a brace, in which the patient left the hospital. Figure 2 b shows the roentgenographic findings eight months after the operation. March, 1935, 21 months postoperative, the patient was allowed to discard the brace; she walked without the slightest limp or pain until six months later, when the patient complained of increasing pain and uneasiness in the right knee joint. Roentgenograms

revealed a slight depression of the median knee joint surface, where most of the pain was concentrated (Fig. 2 c). Since there was the possibility of a pathologic fracture at this site, the patient was operated upon again. From the former incision the bone was exposed; the soft tissues were found to be in intimate connection with the graft; no fracture; no evidence of recurrence was found; just below the insertion of the quadriceps tendon a piece of the cortex was removed in order to inspect the inside of the graft. Necrotic fat tissue and sand-like sequestra were emptied from the superficial spongy layers while deeper inside profuse bleeding was encountered. The upper wire was now removed and the wound closed in layers. After healing of the incision, the patient had



(a) (b)

FIGS. 3a and b.—Same patient as in Figure 2. Two years after operation.

to use the brace again for walking. At the end of 1937 the patient was allowed to discard the brace again. She could walk without any support. A roentgenogram, five and one-half years after the operation, revealed no evidence of recurrence, and no further breakdown of the bony structure of the graft, but roughening of the joint surface (Fig. 2 d). In spite of these changes and a moderate genu varum deformity, the patient walked without any pain or limp. In January, 1939, she felt a dull pain in her epigrastrium and noticed a gradually increasing swelling of both lower extremities and abdomen. Before long she became jaundiced. The family doctor advised against an operation. The patient died, February 19, 1939, five and three-quarter years after the operation. An autopsy was not performed.

COMMENT.—In a 23-year-old patient, the right upper third of the tibia was replaced by a corresponding part taken from a 70-year-old man. Both patients had the same blood group. It must be assumed that the bony parts of this graft died after transplantation—as in any other bone graft. They were gradually regenerated, however, by creeping substitution of the dead bone by new bone. While in autogenous grafts the regenerative process takes

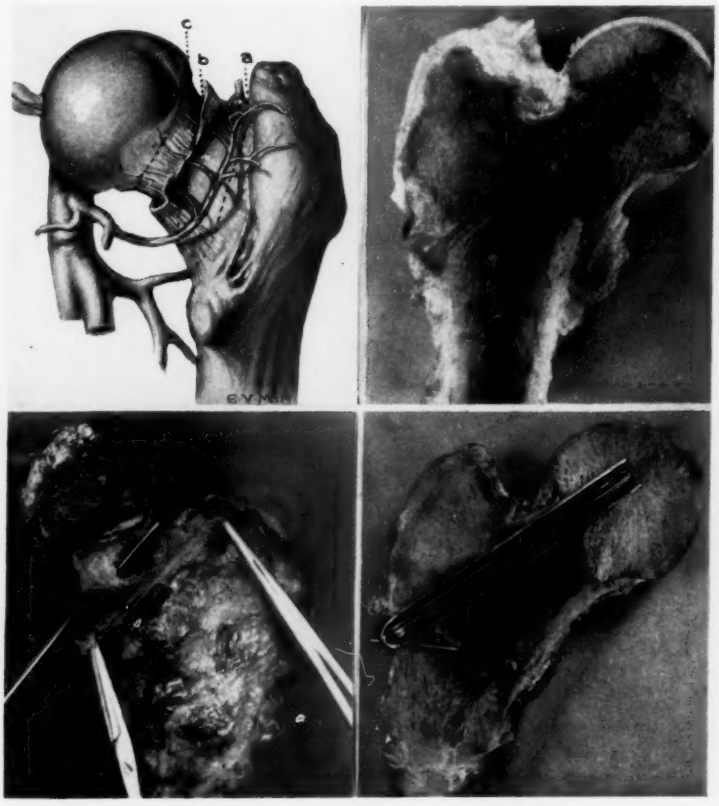
a comparatively short time, in a homologous graft it takes years before the dead bone is substituted by a new bone. In the above case, the greater parts of the graft were still found dead after nearly two years post-operative. No wonder that some parts of the dead cancellous bone broke down upon weight-bearing. It must also be assumed that parts of the articular cartilage had become replaced by fibrocartilage or fibrous tissue, since, according to Phemister, articular cartilage dies if the regeneration of the bone beneath is delayed for longer than one year. Thus homologous half-joint transplantations should be considered only in exceptional cases. Autogenous half-joint transplantations are much more reliable, as clinical and experimental results demonstrate.

REGENERATION OF CERTAIN INTRACAPSULAR FRACTURES

The healing process of an intracapsular fracture of the neck of the femur and certain other intracapsular fractures have often been a matter of dispute; but more light has been thrown recently upon this subject since the regenerative processes in those types of fractures have been compared with those found in bone transplants, more correctly termed half-joint transplants (Axhausen,[2] Phemister[22]). It is generally agreed that in an ordinary fracture the periosteum and endosteum are important healing factors from which most of the callus is derived. Furthermore, in an ordinary fracture both fragments remain alive since the well vascularized periosteum from without and the intra-osseous vessels from within guarantee enough protection. In a true intracapsular (subcapital) fracture of the neck of the femur the fragments have no periosteum, at least not the head. Secondly, it is generally agreed that after an intracapsular fracture the head of the femur dies, at least in most part. The head of the femur receives its main blood supply from intramedullary vessels running through the neck and from capsular arteries. The blood supply through the ligamentum teres, although active in younger individuals (Wolcott,[34] Chandler and Kreuscher[8]), is often inadequate in adults to maintain alone the viability of the head (Kolodny[18]). If a neck of the femur breaks intracapsularly the head of the femur is cut off from its main blood supply. Therefore, the head dies—at least most of it perishes. Such a fragment resembles a bone graft or rather, a transplanted epiphysis. Figure 4 d shows clearly the color difference between the well vascularized neck and the dead head. This femur is an autopsy specimen of an 81-year-old man upon whom I operated two days following the occurrence of the fracture, and who died 16 days postoperative from a cerebral thrombosis. The histologic examination of the head showed many of its osteocytes shrunken, and many empty lacunae. Most of the articular cartilage, however, survived, protected as it was by the synovial fluid within the closed capsule. If the dead parts of such a head fragment become regenerated, the only way possible is by ingrowth of vessels and osteoblasts from the living parts of the neck across the fracture line into the head (Santos[26]) or from the surviving osteoblasts. This process is assured only by complete and prolonged immobilization of both fragments, so that these delicate vessels can cross the fracture line un-

The specimens are autopsy specimens of patients operated on by the author. The pictures were taken with panchromatic and colored film (Kodachrome type A).

(a) (b)



(c) (d)

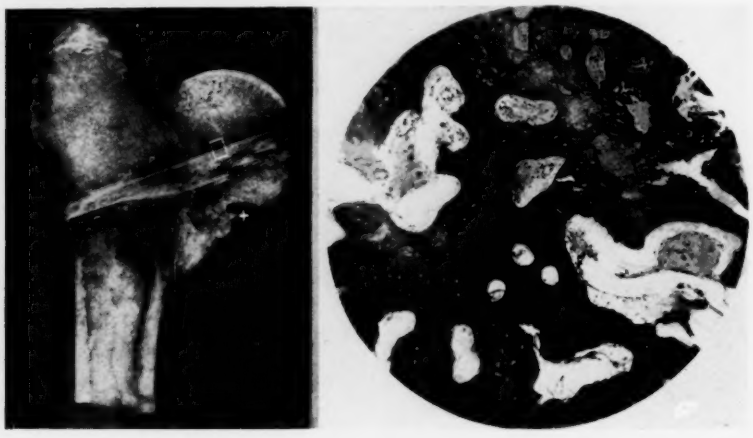
FIG. 4(a).—Vascularization of the neck of the femur comes from three sources; intramedullary vessels, capsular vessels (branches of the art. circumfl. femoris ant. and post.), and from vessels through the lig. teres. The latter source is negligible in older individuals. In a fracture through line (a) both fragments remain vascularized. In a fracture through line (b) the upper half of the head fragment lies intracapsularly, the lower half extracapsularly; head fragment remains partially vascularized. In a fracture through line (c) the entire head fragment lies intracapsularly, becomes separated from its main circulation and is apt to die.

(b). Fracture through base of neck of femur (approximately through line (a) of Fig. 4a). Both fragments remained vascularized (the dark areas are red on the colored film). This fracture had healed in seven weeks. Note the fibrous membrane which surrounded the nail and the marked hyperemia along it (foreign body reaction).

(c). Fracture through neck of femur, approximately through line (b) of Figure 4a. The upper half of the head fragment lies intracapsularly (the upper half of the capsule is severed); the lower half extracapsularly; hence the head fragment remains attached to the main circulation through the lower half of the capsule (note the strong vessel running parallel and close to left border of the capsule).

(d). True intracapsular (subcapital) fracture of neck of femur, 16 days after operation, 18 days after fracture, approximately through line (c) of Figure 4a. The head fragment is separated from its main circulation while the neck is well vascularized (the dark areas are red in the colored film). The head is pale and was found dead when examined microscopically. Note marked hyperemia in the living part of the neck of the femur along the nail; layers of dense fibrous tissue were found along the nail canal in the same section of bone (foreign body reaction); but no such reaction was found in the dead head fragment. Hence a nail membrane developed only in the living part of the bone.

disturbed. Therefore impacted fractures always heal. But even under ideal conditions parts of the head may not become regenerated, particularly peripheral subarticular regions. They may break down on weight-bearing and lead to a deformity comparable with the "Mushroom" deformity of the Osteochondritis Juvenilis Deformans Coxae, a histological picture of which has been described by Perthes Axhausen, Phemister and others (H. May[20]). Hence complete prolonged immobilization by external or internal means is the most important factor in treating the true intracapsular fracture of the neck of the femur. If, due to incomplete immobilization, the vessels are prevented from crossing the fracture line, a nonunion may be the result. The following case may serve as an example:



(a) (b)

FIG. 5a.—Ununited fracture (pseudarthrosis) of the neck of the femur of five years' duration. Note narrow curved fibrous disk running from "+" upward. Section of fibula had been transplanted 10 weeks previously. Marked hyperemia in and around the graft (the dark areas are red on the colored film).

(b). Microphotogram of a section of the fibula—graft and head of the femur (approximately of a region which is marked in Figure 5a). The graft and the host bone are in intimate connection. The graft (lower half of the field), as any other bone graft, has died after the transplantation (note the disappearance of most of the bone nuclei); but there is evidence of transformation of the dead graft into living bone tissue: vascularization of the haversian canals, osteoblastic action particularly in the peripheral parts of the graft. The host bone is alive; its cells contain well stained nuclei; there is marked hyperemia and osteoblastic action. Note the row of osteoblasts lining the medullary space in the center of the field.

Case 3.—A male, age 70, suffered a fracture of the neck of the femur. He stated that his injured leg was placed between two sandsacks; after five weeks he was allowed to sit in a chair, and after eight weeks to walk on crutches. His fracture did not unite; he limped and had pain on walking. Five years after the fracture he consulted me. Because the cleft between the fragments was narrow and, according to the roentgenograms, the head of the femur at least partially alive, a bone grafting operation was advised. I transplanted the middle section of the fibula without its periosteum into the neck of the femur, after Henderson,[14] who uses a guiding wire and cannulated drills to make a proper-sized canal for reception of the fibula graft. The patient developed a postoperative psychosis and died ten weeks after the operation.

COMMENT.—A thorough examination of his femur specimen led to the following conclusion. The original fracture was apparently intracapsular, with the exception of a small part of the head fragment, which remained

attached to the capsule and, thus, to the circulation (Fig. 5 a). It was reasonable to assume that this small attachment was not sufficient to maintain the entire circulation of the head fragment. Circulation through the ligamentum teres was definitely out of the question in this case since it was found detached and shrunken. Hence the greater part of the blood supply to the head fragment was severed leading to partial necrosis of this fragment. Due to incomplete immobilization of the fragments, vessels and osteoblasts could not grow from the alive part of the neck in the partially necrotic head. The result was a fibrous union. To achieve a bony union the fibula transplantation was performed. The grafted fibula died as any other bone graft does after transplantation. Ten weeks after the transplantation, however, marked hyperemia and osteoblastic action could be found in and around the graft both in the section within the neck and within the head (Fig. 5 b). From experience in operations on fractures of the neck of the femur and examinations of various specimens, I am inclined to believe that the true intracapsular (subcapital) fracture of the neck of the femur with necrosis of the entire head, as pictured in Figure 4 d, is the rarer type. It is this type of fracture in which regeneration may be incomplete or entirely absent, causing deformities of the head fragment or nonunion. In most instances of fractures of the neck of the femur smaller or larger sections of the head fragment remain attached to parts of the capsule and thus to the circulation, saving at least some of the head from necrosis. If such a head fragment is attached firmly upon the alive neck fragment by complete immobilization, it has a good chance to regenerate throughout and result in a bony union. The main reason why the internal fixation (nailing, *etc.*) of this type of fracture is more successful than any other treatment lies probably in the fact that it assures better immobilization of the fragments. Even the large plaster encasement, no matter how well-fitting it may be, allows slight movements of the fragments. But internal fixation alone would not be successful were it not for prolonged avoidance of weight-bearing of the affected limb, to protect the dead bone from collapse and to assure immobilization. In extracapsular fractures the fragments are covered by periosteum; second, the intra-osseous circulation of both fragments remains undisturbed (Figs. 4 b and c); therefore, both fragments remain alive and such a fracture heals like any ordinary fracture and as quickly as any ordinary fracture no matter what kind of fracture treatment was employed.

Another similar example is the healing process of a fracture of the carpal scaphoid bone with death of the proximal half followed by bony union. Most of the surface of this bone consists of articular cartilage leaving little room for active osteogenetic periosteum. Hence its viability depends entirely upon intramedullary circulation. From clinical experimental experiences (Watson Jones,[33] Obletz and Halbstein[21]) it is known that in about one-third of the cases the bulk of these vessels enters the bone on the distal (lateral) aspect of the bone and in the constricted midportion leaving the proximal (median) half supplied by the branches of the main vessels so that this part is rather

poorly vascularized (Speed[28]). If, in such a case, a fracture occurs within the proximal half of the scaphoid or through the waist of the bone where the center vessels enter rupturing their trunks, the proximal half of the scaphoid is cut off from its circulation and dies (Fig. 6). It may, however, become revivified by ingrowth of vessels and osteoblasts from the viable distal half (Fig. 7 a–d), provided absolute immobilization was carried out through the time of regeneration.

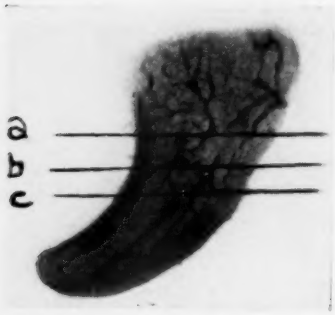


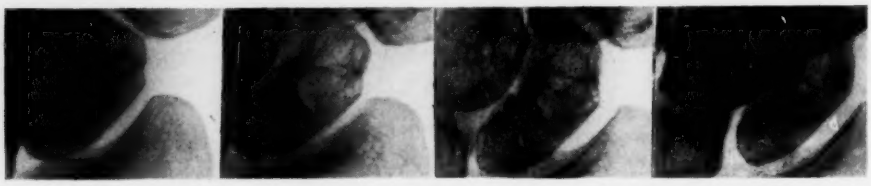
FIG. 6.—Carpal scaphoid bone. Representation of its vessels and the relation between fracture lines and these vessels. (After Böhler and Schnek.[5]) A fracture through (a) will keep both fragments alive while a fracture through (b) and (c) will cause a necrosis of the median (lower) fragment.

This type of regeneration apparently occurs in any intracapsular fracture in which one fragment has become separated from its circulation, followed by bony union.

CONCLUSION.—Certain intracapsular fractures do not heal like ordinary fractures. They heal in a manner similar to the regeneration of a transplanted epiphysis; namely, rather by bone regeneration and osteoconduction than by osteoproduction. The articular cartilage, however, remains alive, provided there is no delay in regeneration of the bony constituents (Campbell,[7] Phemister[22]).

SUMMARY

The processes involved in regeneration of joint transplants were discussed. These biologic processes were paralleled with those found in certain intracapsular fragments such as the head of the femur after a subcapital fracture and the median third of the scaphoid bone after certain fractures. A case of a fracture-dislocation of the humerus was described in which the head of the humerus was reimplanted after temporary removal (autogenous half-joint transplantation). Another case of a 23-year-old patient was described in whom the upper third of the tibia, including the joint surface, was removed and replaced by a corresponding part of the tibia obtained from a 70-year-old man (homologous joint transplantation).



(a) (b) (c) (d)

FIG. 7a.—Fracture of right carpal scaphoid bone (approximately through line (c) of Figure 8).
(b). Same fracture two months later. The distal (lateral) fragment is alive; the proximal (median) fragment is dead. The distal fragment had atrophied together with the other bones; the proximal fragment has remained dense because its calcium could not be carried away due to lack of vessels.
(c). Four months later the proximal fragment presumably has become revivified.
(d). Six months later the fracture has healed.

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BRIEF COMMUNICATIONS

RAYABLE GAUZE*

DEVELOPMENT OF A RADIOPAQUE, THREADED GAUZE SURGICAL SPONGE

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"With vivid words these just conclusions grace,
Much truth compressing in a narrow space;
Then many shall peruse, but few complain
And envy frown, and critics snarl in vain."
—PINDAR.

IT IS undoubtedly true that human interest is centered more on *what* has been done than on *how* it has been done. Thus, the following short summary is drawn from long experimental studies, and is offered to make operative surgery safer for the patient.

Present methods and devices of sponge control have, thus far, failed to provide entire security against the accidental havoc of the "lost" sponge. Preventive measures and safety arrangements have been undone by the frailty and error within the human equation.

TABLE I

PREVENTIVE MEASURES DEVISED FOR SPONGE CONTROL

(*After Crossen and Crossen*[6])

1. SPECIAL ATTENTION
   - BY OPERATING TEAM
   - AIDS TO ATTENTION
2. COUNTING SPONGES
   - PRE-CLOSURE COUNT
   - POST-CLOSURE COUNT
   - AIDS TO COUNTING
3. ATTACHING TRACERS
   - TAPE TO SPONGE
4. MISC. EXPEDIENTS
   - STICK SPONGES
5. CONTINUOUS SPONGE
6. RAYABLE GAUZE
   - METAL RING
   - WIRE THREAD
   - CHEMICAL IMPREGNATION
   - FIBERGLAS THREAD

A specially prepared radiopaque thread is presented which embodies the qualities of marked radiopacity, relative chemical, physical and biologic inactivity, softness comparable to silk, and nominal cost of production.

It is suggested that in the manufacture of all surgical gauze that is to be used in the operating room, a single strand of lead "fiberglas" thread might be incorporated into the gauze mesh, or so placed in prepared pads, sponges and gauze drain filler, that "lost" sponges can be more readily retrieved by roentgenographic identification.

Although such a radiopaque surgical gauze would not guarantee security against the losing of a sponge, by pointing to its presence and location it would mitigate the otherwise likely consequences of such an accident.

* Read before the Baltimore City Medical Society, Baltimore, Md., January 3, 1941.

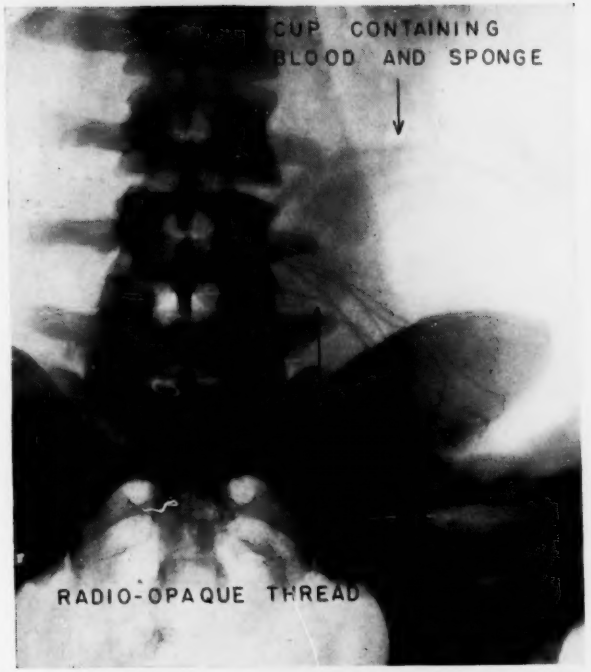

FIG. 1.—A "rayable" sponge placed in a cardboard cup containing 250 cc. of citrated blood; and an abdominal film taken through an average-sized person. The shadow cast indicates that blood cannot "black-out" the opaque thread.

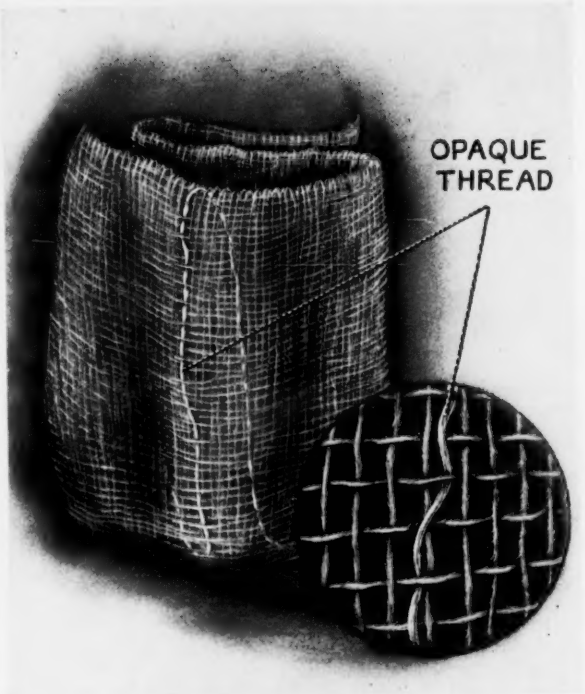

FIG. 2.—An artist's impression of "rayable" gauze. A single strand of thread is all that is necessary.

FIG. 4.—Roentgenogram of the right upper quadrant of an obese female. The "fiberglas" thread is contrasted with residual barium in the large bowel and a gallstone.

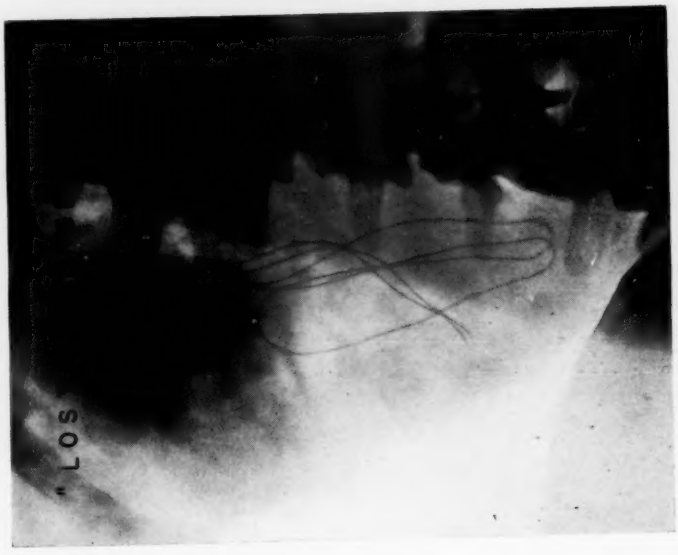

FIG. 3.—The elimination of the gauze foreign body hazard in pelvic and abdominal surgery can be accomplished by the use of a radiopaque threaded pad. The thread is clearly defined against the skeletal structures.

Rayable gauze fulfills the standards set by the American College of Surgeons, is applicable in every surgical situation, and is a comparatively inexpensive premium to pay for operating room security.

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AN ATRAUMATIC FASCIAL-SUTURE NEEDLE

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IN 1901, L. L. McArthur[1] wrote his first article on a method of repairing herniae by the use of "living" fascial sutures. His described technic consists of splitting from the edge of the internal flap of the external oblique aponeurosis a one-eighth to three-sixteenths-inch strip of the white fibers which enter into the formation of the internal pillar of the ring. The strip is cut loose from its insertion in the muscle belly, but left attached at its inferior end to the spine of the pubis. A similar strip is taken from the outer flap, the lower end of which strip terminates in the fibers of the external pillar of the ring.

The operation is then completed according to the choice of the operator, either a Bassini or an Andrews procedure, using these strips as suture material for a running stitch.

His reasons for using his "tendon suture" were: (1) Obtaining of a "living" suture which lies directly in the wound itself instead of requiring extensive dissections or abnormal displacements of important structures; (2) lessening the chance of failure through avoidance of introduction of dead or foreign tissue; (3) healing of the tissue *in situ*, rarely sloughing or becoming absorbed; and (4) incorporating of organized white fibrous tissue into the resisting cicatrix of the healed wound. When the fascial strips are loosened they roll up as does catgut, pull through as easily as catgut, and have a tensile strength of 11 to 24 pounds. He felt that the same procedure could be applied to other situations.

McArthur's technic gained little support from his contemporaries, and was nearly lost in the mass of medical literature. It was not until about 20 years later that the use of fascial sutures was revived. In 1921, Gallie[2] became interested in the large number of ventral herniae found in soldiers who had been operated upon for abdominal wounds incurred during World War I, and in recurrent herniae of other types. He noted that, in most instances, the muscular and aponeurotic structures which had been sewn together in an attempt to close abdominal wall defects, due to extensive war injuries, had not remained firmly healed together, but had separated sufficiently to give rise to ventral herniae. Thus, he was led to the idea of closing these defects in the abdominal wall with free transplants of fascia lata strips woven into what he called an "aponeurotic filigree." He then demonstrated that these strips, when placed in such a position that they received an adequate supply of lymph, continued to live unchanged.

After conventional suture operation for inguinal hernia with recurrences, in no case did Gallie find any evidence of adhesion between the conjoined tendon and the inguinal ligament, stating that: "From our clinical experience, therefore, and from the information derived from our experience, we have come to the conclusion that operations designed to produce permanent adhesion between aponeurotic structures having a natural tendency to separate are defective in principle, if they depend solely upon the process of healing in the line of suture. Herein lies the explanation of the high percentage of recur-

rences in large ventral herniae and in indirect inguinal herniae, even when the operations have been performed by the most competent hands." Therefore, Gallie began using transplants of fascia lata as living sutures which might permanently hold the edges of hernial rings together without depending upon the natural process of repair. Gallie, using fascia lata strips, corroborated McArthur's findings that a living suture had the great advantage over catgut and similar sutures, in that it was not absorbed but continued to perform the function for which it was originally intended. Over silk and other nonabsorbable sutures it was superior in that it, being composed of a perfectly nonirritant

FIG. 1.—Head of the needle unscrewed; note the gaping jaws. (×2)

FIG. 2.—Head partially screwed into the shaft. Jaws now partially closed. (×2)

living tissue, healed solidly into the tissues through which it passed. He, furthermore, demonstrated that it did not show a tendency to cut-out when subjected to any normal physiologic strain.

One of the chief technical difficulties in the use of fascial sutures has been the lack of a suitable needle to draw the fascial strip through the tissues with the least possible trauma. McArthur tied a strand of No. 3 silk (with a single knot) tightly to the free end of the strip of fascia, threaded the silk into an ordinary needle, and then pulled the fascial suture through the tissues to be united. In using this technic it is apparent that the operator would encounter difficulty in drawing the suture through so firm a structure as the aponeurosis of the external oblique, or through the inguinal ligament.

Gallie threaded the fascial suture into a needle with a large eye (so-called Gallie needle). He then tied the strip of fascia securely into the needle with catgut or silk to prevent its unthreading, and tied a ligature of catgut around the terminal end to prevent it from splitting. In taking the first stitch, he recommended that the needle be passed through one of the edges of the gap to be closed and through the terminal end of the suture, and drawn taut—thus producing a sort of slip-knot to anchor the fascial strip. He ended the suture by splitting the terminal end, and tying the two strands thus produced about itself in a triple knot. This knot was made secure by transfixing it with a catgut ligature, to hold the loops together until they became firmly healed.

Gallie's technic of attaching the fascial suture into a large-eyed needle was

an improvement in that the semicutting-edged curved needle started a small rent in the aponeurosis through which the suture could be pulled. But it is obvious that the fascial suture was, of necessity, of double thickness at the point where it was threaded through the eye, and that this double thickness produced undue trauma when it was forced through the tissues.

In an attempt to overcome this difficulty I devised a comparatively atraumatic needle. Basically, it is a semicutting-edged, curved, two-pieced needle of approximately the same size as the so-called Gallie needle. The distal section, about three-quarters the length of the entire needle, consists of a triangular semicutting edge on a curved shaft. At the proximal end of this section is a female thread which has been longitudinally countersunk to a depth of about one-quarter inch, the countersinking being of the greatest possible diameter consistent with the strength of the spring steel shaft. Into this screws the short proximal section which, manufactured out of a specially tempered spring steel, is of the same diameter as the rest of the needle, except that its proximal two-thirds is hollowly slit longitudinally down the middle, and its distal half tapers down to a male screw of much less diameter. When unscrewed, this section gapes open; but when screwed into the shaft of the distal section the tapering fits tightly into the countersinking and the gaping ends are forced together.

In use, the end of the fascial suture is placed between the gaping ends of the head of the needle, and the head screwed into the shaft. The squeezing pressure of the split ends coming together retains the suture in the needle. As a further aid to holding the suture in place, a tiny pin is incorporated at right angles into the medial surface of one side of the split. This pin digs at right angles into the suture when the head is tightened.

The result is a streamlined needle-fascial-suture junction. The fascial suture may now be pulled through the fibrous tissues of the aponeurosis, inguinal ligament, and conjoined tendon with no more trauma than that which is caused by the needle alone. The diameter of this needle is about three-sixteenths inch, just large enough to be attached to the most commonly used fascial suture strips which are one-eighth to three-sixteenths inch in width.

This needle has another advantage, in that it can be used repeatedly. Furthermore, it may also be attached to kangaroo tendon sutures, ribbon-gut, and for suture materials of similar types.

SUMMARY

(1) A brief résumé of the history and uses of strips of living fascia as suture material has been given.

(2) Some of the advantages of fascial sutures over conventional sutures in the repair of various types of herniae have been described.

(3) The author has described an atraumatic needle of simple design, which may be used to facilitate the use of fascial and other large caliber sutures.

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A PUNCH FOR CUTTING COTTONOID SPONGES FOR USE IN NEUROSURGICAL OPERATIONS

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GENTLE MANIPULATION of nervous tissue is of even greater importance in neurosurgical procedures than in most other types of surgery, because irreparable injury to the brain or spinal cord may be easily produced. Circular cottonoid pledgets and rectangular strips or some other nontraumatizing material are indispensable for sponging nerve tissue and for protection under retractors. As sponges, the moist, circular pledgets readily absorb blood and cerebrospinal fluid, due to enormous capillary surface afforded by the fibrils. Application of the suction tip directly to the pledget overlying the bloody surface rather than directly to the tissue prevents injury by the suction tip and facilitates suction by obviating clogging of the instrument. In addition, the capillarity of the pledglet is effective in permitting the entire field it covers to be cleansed, although the suction tip is applied to but one point on the pledget.

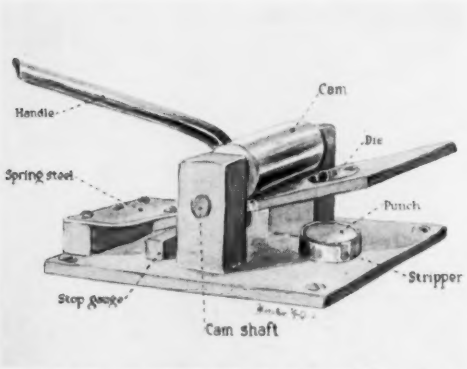

FIG. 1.—View of punch showing relation of cam shaft to cam when not in operation. The handle is pictured shortened in order to conserve space.

In the long, painstaking procedures involved in such operations as brain tumor removals, over 200 circular pledgets may be used. These are usually of two diameters: (1) About three-quarters of an inch; and (2) about one-half or three-eighths of an inch. Cutting these out by means of scissors is a tedious and tiring procedure.

In the operating rooms at the University of Minnesota Hospitals, two punches for cutting the circular cottonoid pledgets are in regular use—one cutting smaller, the other cutting larger sponges. Both punches, one of which is shown in Figure 1, are mounted on a single, heavy iron casting, for stability during stamping. The punch assembly consists of the circular punch proper, mounted on a base 4½x4x½ inches, a die plate mounted on a flap of spring steel, and a cam actuating the die plate and operated by a handle about ten inches long. The cam shaft is eccentrically mounted, permitting gradual downward movement of the die on the punch as the arc described by the handle increases. An iron stop-gauge limits the descent of the die plate. A segment of thick rubber tubing slipped over the punch and allowed to project slightly above it serves as a stripper.

The exact dimensions of the apparatus are indicated in the work diagrams Figures 2A and 2B. Figure 2A is a side elevation and Figure 2B is a front

elevation, the die plate (D.Pl.) and punch being shown engaged with the stripper compressed. In this position, the handle is nearly parallel to the base. The cam shaft mounting requires no lubrication, but friction between

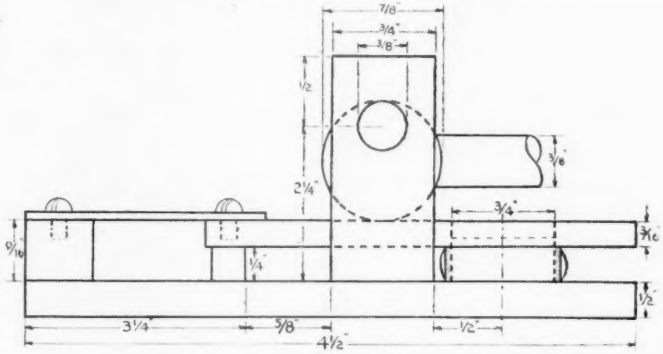

FIG. 2A.

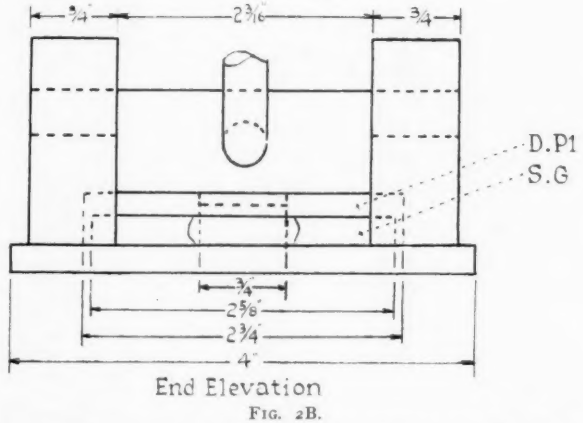

FIG. 2B.

FIG. 2.—Work diagrams of punch in stamping position. (A) Side elevation. (B) End elevation. D.Pl. die plate. S.G. stop-gauge.

the cam and the die plate can be minimized by a light coating of machine oil. The moving parts of the apparatus are sturdy, requiring no replacement. The punch and die are made of high carbon steel so that necessity for sharpening is eliminated. A single punch assembly can be made for approximately seven or eight dollars.

In operation, a large sheet of cottonoid is passed between the punch and die plate, and pledgets are stamped out from each fresh area, at the rate of about 60 per minute.

SUMMARY

An inexpensive punch is described for stamping out circular cottonoid pledgets used as sponges in neurologic surgery. The ease and rapidity of operation results, primarily, from a minimum of movable parts.

Appreciation is herewith extended to Mr. John A. Phelan of the University of Minnesota Scientific Apparatus Shop, for his wholehearted cooperation in the construction of this punch and preliminary experimental devices.

BOOK REVIEW

A MANUAL OF THE TREATMENT OF FRACTURES. By John A. Caldwell, M.D. Charles C. Thomas Co., 1941.

A Manual of the Treatment of Fractures, written in a plain and simple text, by a Professor of Clinical Surgery, for students and interns, and dedicated to the House Officers of the Cincinnati General Hospital, is offered by the publishers.

With such a clear statement of its objectives, this manual should create an unusual amount of interest among such groups, and to a far greater number of men represented by the general practitioners.

The need for such a Manual has long been recognized, and this contribution of Dr. Caldwell's meets it, as would be expected from such a master surgeon. The fact that the author is a general surgeon, and Director of the Fracture Service of the Cincinnati General Hospital, qualifies him to speak upon this subject.

His experience is evidenced throughout the text by such statements as:

"The use of local infiltration anesthesia for the reduction of fractures."

"The most useful form of splinting material is plaster of paris."

"It is always a serious mistake to administer an anodyne to relieve pain or discomfort caused by splints."

"Ascertain beforehand if the patient is serum sensitive before giving antitetanic or other forms of sera."

His reference to the Orr treatment in compound fractures instead of the more complicated technic of Dakin-Carrel, is now generally accepted.

The use of line drawings for illustrations instead of the usual reproduction of roentgenograms and photographs is an innovation which should be commended.

It is unnecessary to say that such a book is timely, in the face of the increasing demands now being made by military, industrial and automobile accidents.

WALTER ESTELL LEE, M.D.

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